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PROCEEDINGS

OF THE

NEW YORK

PATHOLOGICAL SOCIETY

FOR THE YEAR

1888

PRINTED FOR THE SOCIETY

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PROCEEDINGS

OF THE

NEW YORK PATHOLOGICAL SOCIETY.

Stated Meeting January 25, 1888.

W. P. Northrup, M.D., President, in the Chair.

Dr. W. B. James presented a series of microscopical preparations of blood taken from patients suffering from malarial fever, which demonstrated the various forms of the

MICRO-ORGANISMS OF MALARIA.

The attempts to discover the cause of malarial disease are of considerable historical interest. From earliest times the existence of a contagium vivum has been generally assumed, by reason of the peculiar clinical features of the disease itself. Up to 1879 most of the hypotheses that were offered assumed that some one of the fresh-water algæ was the exciting cause.[1] These observations were never confirmed, and obtained but little belief.

In 1879, Klebs and Tommasi-Crudeli [2] isolated from the soil of the Roman Campagna a bacillus which they believed to be the cause of the malarial diseases existing in that neighborhood. They obtained pure cultures of the organism, and stated that injection of these cultures into rabbits was followed by typical intermittent fever, with organic lesions similar to those found in persons dying of malarial disease. They gave to the organism the name bacillus malariæ. Their results were not confirmed by subsequent observers. It is now generally believed that the so-called bacillus malariæ has no connection with the disease. Yet, until Laveran published his paper on the so-called plasmodium malariæ, the work of Klebs and Tommasi-Crudeli was the most important attempt that had been made to discover and define the cause of intermittent fever.

The first work on the plasmodium malariæ was published in 1881 by Laveran,[3] a French army-surgeon in Algiers. He discovered and described, in the blood of his

cases, certain appearances that had never previously been noted. He offered a new interpretation of other phenomena that had long been recognized in malarial blood, but not before explained. To account for all the phenomena observed, he assumed the presence in the blood of a parasite, a polymorphic organism belonging to the animal kingdom.

Since Laveran's paper, much work has been done on this subject, though not a great deal has been accomplished beyond a confirmation of his statements. In Italy, Marchiafava and Celli [4] have devoted much time to the study of this question. In this country, Councilman [5] and Osler [6] and Sternberg have studied it most. At present,

a, Unpigmented amœboid body; *b*, pigmented amœboid body; *c*, encysted stage; *d*, encysted form, granules collecting in centre; *e*, segmenting body; *f*, free spores and pigment; *g*, *h*, crescentic bodies; *i*, flagellated body; *k*, normal red blood-corpuscle.

our knowledge of the organism may be briefly summed up as follows: In the blood of persons suffering from malarial disease there is a series of phenomena not yet found under any other conditions. These phenomena point to the presence of an animal parasite. The appearances that we get are one or more of the following:

First.—Colorless protoplasmic bodies inside the red blood-corpuscles. They vary in side from one-fifth to almost the whole diameter of the corpuscle. They exhibit active amœboid movements. Some contain scattered granules of brownish-black pigment; others are unpigmented. The red corpuscle which contains the amœboid body is commonly larger, and paler than normal.

Second.—Disk-shaped bodies of colorless protoplasm. They are somewhat larger than a red blood-corpuscle.

They show no amœboid movements. They contain scattered pigment-granules. They are apparently a later stage of the form first mentioned, which has come to occupy the entire corpuscle, and has then entered upon a cyst stage.

Third.—Forms similar to the cysts, but in which the pigment-granules have become massed at the centre, while the protoplasm is undergoing segmentation. Then there are found various stages in the transition from the encysted to the segmentary form.

Fourth.—Small masses of protoplasm that have resulted from the segmentation of the cyst form. They are commonly somewhat oval in shape. In fresh blood it is difficult or impossible to distinguish them from blood-plaques. In dried blood they show a tolerably characteristic bipolar staining with aniline dyes.

Fifth.—Hyaline bodies of crescentic shape, in length rather more than the diameter of a red blood-cell. They have, in every case, a collection of pigment-granules in their centre. These are the so-called "crescentic bodies."

In some cases there are found bodies resembling the crescents closely in character, but elliptical or round. The crescents may have a delicate curved outline opposite the concavity or convexity, or both, but this outline never reaches quite to the tips of the crescent.

Lastly, there are the various motile bodies. These are: First, flagellated bodies. A round or pear-shaped body about one-half the diameter of a red blood-cell. It contains pigment-granules, and is provided with from one to four flagella, which show an active lashing motion, and by means of which the organism moves about in the blood-plasma. The flagella are several times as long as the diameter of a red cell. Their motion is active enough to set up well-marked movements on the part of the neighboring blood-corpuscles.

Secondly, free flagella have been described. They seem to have become detached from their bodies, and to be capable of leading an independent existence. They exhibit active movements.

Thirdly, some observers have described hyaline pigmented bodies with an actively moving, undulating periphery.

Pigmented white blood-corpuscles have been described. I have not seen them; nor have I in my cases met with free flagella or the bodies with undulating periphery. All of the other forms I have had frequent opportunity to study.

Next, as regards the interpretation of the appearances described in the blood: It is believed that the unpigmented amœboid body makes its way into a red blood-

corpuscle, and lives and increases in size at the expense of the latter. It becomes pigmented, the pigment-granules being changed hæmoglobin. It has been demonstrated that the granules contain iron.

Having come to occupy the entire, or almost the entire, corpuscle, it enters upon a cyst stage, during which the pigment-granules make their way to the centre, and segmentation of the periphery takes place, thus forming spores, which are subsequently set free. Each spore then begins again the same life-circle.

The biological relation of the crescentic and flagellated forms to the other has not been made out. But there are, among the lower animal organisms, many polymorphic varieties, which at different periods of development exist as spores, amœboid bodies, cysts, and flagellated organisms. Among the protozoa, some forms (sporozoa, Leuckart) [7] develop falciform spores which in appearance resemble the crescents.

As regards the relation of the various forms of the organism to the types and periods of the disease: The description that I have given applies to tertian intermittent. With quartan I have had no experience; but Golgi [8] states that in this form, which is the prevailing type in Pavia during the autumn months, the organism exhibits certain peculiarities. In general, the various forms are more clearly marked off from one another. The segmentation forms are especially clear. He also states that in every case the complete development of the organism corresponds to the interval between two paroxysms of the disease.

The paroxysm seems to be the period at which the segmentation and setting free of spores takes place. This is now tolerably definitely settled. The discovery in the blood of the segmenting bodies justifies the positive prediction that the patient will have a chill at some time within two hours. According to Golgi, immediately after the paroxysm one finds free spores and unpigmented amœboid bodies. During the day of intermission the pigmented amœboid forms gradually increase in size till six or eight hours before the chill, when the cyst stage is met with. Immediately before and during the chill one finds the segmenting forms.

In my own cases I have found the segmenting forms always just before and during the paroxysm, and at no other time. I have, however, found the pigmented amœboid bodies at all times, and most numerous during the paroxysm.

It is said that in some epidemics the organisms remain unpigmented throughout the disease.[9]

The crescents and the bodies allied to them occur in

chronic cases, usually in those associated with malarial cachexia. This point is one upon which all observers agree.

In two of my cases there were at the same time crescents and amœboid forms. Both of these forms commonly occur in greater number in severe than in light cases, though to this there seem to be exceptions. The various flagellated forms may occur in any case; but in only a small number of cases in blood from the finger. I found them in only three of my cases.

During the past autumn I had an opportunity to study the blood of thirty-five cases which were undoubtedly malarial disease. In thirty-four of the cases I found the hæmatozoon. In one case the examination was negative, but here I was able to examine the blood only on one occasion. Often, when the number of the organisms in the blood is small, one may look through several slides before finding them.

In my cases, as in those published by others, the amœboid forms, pigmented and unpigmented, occurred in the acute stages of the disease; the crescentic forms, in the chronic stages. In no case have I failed to find the organism, where the patient presented a clear clinical picture of malarial disease, and where I was able to make a number of examinations of the blood.

The examination for the organism has considerable diagnostic value. In several cases I have been able to diagnose or to exclude malarial disease by examining the blood, where the symptoms alone were not characteristic enough to make a positive conclusion possible, and where the subsequent history has borne out the results of the microscopic examination.

The microscopic technique is simple. The blood is best examined fresh, spread out in the thinnest possible layer —the rouleaux of corpuscles broken up. High-power objectives are necessary. A $\frac{1}{12}$-inch oil immersion answers well.

Stained specimens are best prepared by drying the blood in a very thin layer on a cover-glass, which is then passed through a flame, as in examining for bacteria. It may be stained in a watery solution of an aniline dye, washed out in water, or partly decolorized in alcohol, and mounted, as usual, in balsam. Fuchsin and methylene blue give the best results. I find methylene blue most satisfactory, the red corpuscles staining light green, and the hæmatozoa blue.

Unpigmented amœboid bodies are best studied in stained specimens. The pigmented amœboid bodies, which are those most commonly met with, are best seen in fresh blood. The crescents also show most satisfactorily un-

stained. The segmenting bodies and free spores are best studied after drying and staining.

The apparent effect of medication on the organism is important.

A few large doses of quinine are almost invariably followed by a disappearance from the blood of the various amœboid forms. The crescentic bodies remain unchanged after quinine, but commonly show a diminution in number as the health improves under the treatment proper for chronic paludism.

These apparent results of treatment followed in my cases as in those of most other observers. The organism has not yet been discovered except in human blood and organs. It has never been isolated. It has never been cultivated outside the body.

Inoculation experiments on human beings are almost uniformly successful. Intravenous injection of malarial blood into a healthy individual is followed by typical intermittent fever, with the appearance in the blood of the second person of the various forms of the organism described. These experiments have been made by Gerhardt,[10] in Germany, and by Marchiafava and Celli[11] and other Italians.[12]

Subcutaneous injections have been unsuccessful.

The few inoculation experiments that have been made upon monkeys have been unsuccessful.

As regards the name of the organism: The term plasmodium has been improperly used in this connection. This name has long been applied to a segregation stage of some of the mycetozoa,[13] and so means, not a particular organism, but a stage of development common to many different organisms. Moreover, so far as we know, the malarial germ has no plasmodium stage.

It is much better, then, in designating the malarial organism, to use the term hæmatozoon of malaria, as suggested by Laveran, which commits us to no definite classification.

Mosso[14] injected into the peritoneal cavity of pigeons and chickens blood taken from the carotid of a dog. After two or three days he killed the former, and found in their abdominal cavities a fluid, the remains of the injected blood. This contained some red blood-corpuscles of the bird and some of those injected from the dog. The latter, he claims, showed appearances identical with those described as the hæmatozoon of malaria. Mosso argues from this that the so-called hæmatozoon is not an independent organism, but merely a metamorphosis of the protoplasm of red blood-cells. Marchiafava and Celli have repeated Mosso's experiments with negative results. Maragliano[15] studied the behavior of healthy human red blood-cells

when subjected to various influences. He kept for some time slides of blood the evaporation of serum from which was prevented. Other slides he subjected to pressure, others to heat. He says that under these circumstances he obtained in the red corpuscles amœboid bodies and segmenting bodies identical in appearance with those described as the organism of malaria. In blood impoverished from any cause he finds these appearances more pronounced. He does not state whether he has or has not himself studied malarial blood. I have repeated his experiments with healthy and impoverished red blood-cells, and have seen appearances which correspond fairly closely with what he has described. But in no case have I seen anything bearing close enough resemblance to the so-called hæmatozoon of malaria to justify the belief that they are identical. Klebs [14] states that Rosenstein found in the blood in typhoid fever appearances similar to the so-called malarial organism; that Dujardin found them also in healthy blood, when evaporation of serum was for a time hindered; that Hoffmann saw such appearances in pernicious anæmia.

Pfeiffer [16] claims to have found similar phenomena in the blood in scarlet fever, mumps, and in vaccination.

It has been suggested, too, by others that perhaps these appearances may mean merely a retrograde change in the red cells, and may accompany any of the diseases to which marked organic blood-changes belong.

As a contribution toward the settlement of this point, I have undertaken the examination of the blood in a series of cases of other diseases where a malarial element might fairly be excluded. In every case I have used either a one-eighteenth or one-twelfth oil-immersion objective and the same precautions, as in the examination of malarial blood. The cases examined were 76 in number, and were as follows: Chronic phthisis, 11; pernicious anæmia, 3; chlorosis, 14; leucocythæmia, 3; jaundice, 3; cancerous cachexia, 3; chronic lead-poisoning, 1; typhoid fever 10; pneumonia, 4; diabetes mellitus, 2; anæmia from cardiac disease, 2; acute anæmia following hemorrhage, 4; acute articular rheumatism, 5; cirrhosis of liver, 1; septic fever, 2; chronic Bright's disease, 5; scarlet fever, 3.

In the above cases I found in the blood no appearances that could be confounded with those in malarial blood by one accustomed to examine the latter. There were in the red cells only those changes usually found in blood impoverished from any cause.

As regards the classification of the organism, we place it in the animal kingdom—morphologically, because it is at no time enclosed in a cell-wall of cellulose; physiologically, because it seems incapable of making its protein

from simpler substances. It seems at all times to be unicellular, so we call it a protozoon.

Further positive classification is at present unsafe. It seems, however, from its general characteristics, to belong among the flagellated infusoria. In connection with the hæmatozoon of malaria the consideration of the other parasitic protozoa is of interest. The study of these has of late been neglected, and the difficulties in the way of investigation, and especially of cultivation, of these organisms, have put rather narrow limits to our knowledge of them. Cartulis [17] has recently found, in the stools of Egyptian dysentery, and in pus from abscesses of the liver, in cases of dysentery, large amœbæ which, he believes, have a causative relation to the disease. He has found them in every case of the dysentery, and never under other conditions. He has not worked out their life-history. His work has not been repeated by others.

Deichler [18] has described protozoa in the expectoration of persons with whooping-cough. Pfeiffer and Loeff [19] have very lately described a most interesting series of amœboid organisms, protozoa, existing in the contents of small-pox, varicella, and vaccinia pustules. But the parasitic protozoa of the blood bear an even closer relation to the malarial question.

A peculiar organism [20] has long been known in the blood of many frogs. It was supposed by Gaulé to be merely a metamorphosis of the protoplasm of the red cells. It is often called "Gaulé's Wurmchen." Ray Lankester [21] and others have proved that it is an independent organism—a protozoon. Part of its life is spent in the interior of a red blood-corpuscle.

Wittich [22] has found in the blood of moles protozoa resembling closely those in malarial disease in man.

Marchiafava and Celli,[11] examining the blood of frogs living in ponds about Rome, saw hæmatozoa very like those in malaria. In one case they found a flagellated organism which lost its flagella, these latter then going on to lead an independent existence.

Mitrophanow [23] and Danilewsky [24] find similar flagellated protozoa in the blood of fishes, especially the carp and mud-fish; also in various other lower animals—frogs, turtles, lizards, and others.

Lewis [25] and Crookshank, in India and in London, find similar organisms in the blood of apparently healthy rats.

It is interesting to notice that, in the above list of animals, most are more or less continuously subjected to the influences and environment that in human beings are popularly supposed to give rise to malarial disease.

The above-mentioned parasites are usually called physiological hæmatozoa, since the animals serving as hosts

seem healthy. It is very possible, though, that they may be the cause of diseases on the part of the host, clinical evidence of which we are unable to discover. The question whether certain fish, lizards, and birds are perfectly healthy or not is one difficult or impossible to settle.

The only apparently pathogenic protozoon that has been studied in the lower animals is that of "surra." Surra is a disease occurring in India in horses, mules, and camels. It has a distinctly intermittent character, and is accompanied by high fever, jaundice, and prostration. It is commonly fatal. In the blood of the diseased animals, Evans, Steele, and Crookshank[26] have uniformly found a flagellated protozoon which seems to be polymorphic. The organism disappears from the blood during the intermission, to reappear again during the paroxysm.

The intravenous injection of blood from a diseased into a healthy animal is followed by the disease in typical form, and by the appearance of the hæmatozoa in the blood of the second animal. The organisms are more numerous in severe than in mild cases. They are very like those found in the blood of apparently healthy rats. They have not been cultivated outside the body.

Many of the lower fresh-water plants are infested by parasites, which show striking points of similarity to the hæmatozoa of malaria. These are actively moving flagellated bodies which find their way into a vegetable cell. The body loses its flagellum and becomes amœboid. It lives at the expense of the cell it occupies, increases in size, and stores up within itself granules of brownish-black pigment which are changed chlorophyl.

Having completed its amœboid stage, it becomes encysted. The pigment-granules are gradually massed at the centre, and segmentation of the peripheral protoplasm takes place. The segments are spores, usually swarm spores, which are set free and again begin the same life-history.

These organisms were well studied by De Bary[27] and Zopt[28] long before Laveran's first work on the malarial organism was published. They are classed among the mycetozoa or myxomycetes. Each parasite is, as a rule, peculiar to some one plant, usually one of the fresh-water algæ.

The points of marked similarity between the mycetozoa and the hæmatozoa of malaria suggest a possible genetic connection between them, and that the malarial organism may exist outside the body as a parasite of some plant.

If malarial disease is caused by a micro-organism, we must assume that it is capable of indefinite life and multiplication outside the body. Only this supposition would explain the phenomena of intermittent fever. Chlorophyl

seems to stand in closer relation to hæmoglobin than any other substance found in the inorganic and plant world.

It seems possible, then, that the malarial hæmatozoon may be also a plant parasite, and that the distribution of malarial fevers may depend upon botanical conditions, or the presence in a given locality of a plant capable of serving as host for the organism.

In conclusion, then, we may state as facts:

1. The presence of the phenomena grouped under the name hæmatozoon of malaria in the blood of persons suffering from malarial disease.

2. The presence of crescentic bodies, only in chronic cases.

3. The presence of the segmenting forms only immediately before and during the chill.

4. The disappearance of all but the crescentic forms after the administration of large doses of quinine.

5. The possibility of transferring malarial disease from person to person by intravenous injection of malarial blood.

But we must at present recognize as hypothetical, though probable:

1. An etiological relation between the appearances described in the blood and the disease itself.

2. The absence of these appearances from the blood in all other conditions.

3. The assumption that the bodies described are one and the same organism.

4. The assumption that any of the forms excepting the flagellated bodies are independent organisms.

Finally, I wish to express my thanks to Dr. Francis Delafield and Dr. William H. Draper, and to the house staff of Roosevelt Hospital, for their kindness in allowing me access to the cases in the wards of the Roosevelt Hospital, and to Dr. Councilman, of Baltimore, for many valuable suggestions in regard to the study of this subject.

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[1] Mitchell: On the Cryptogamous Origin of Malaria. Philadelphia, 1849. Salisbury: American Journal of the Medical Sciences, January, 1866. Hanon: Jour. de Méd. de Bruxelles, May, 1866. Corput: Jour. de Méd. de Bruxelles, March, 1866. Balestra: Richerche ed Experementi, etc. Rome, 1868. Schurtz: Arch. f. Heilkunde, 1868, S. 69.

[2] Klebs and Tommasi-Crudeli: Arch. f. Exper. Pathologie. Bd. xi., S. 122 und 311. Die Malaria in Rome. Munich, 1882. Tommasi-Crudeli: Rendiconti della R. Accademia dei Lincei di Roma, vol. ii., 1886.

[3] Laveran: L'Union Méd., 1881, 95. Traité des Fièvres palustres, Paris, 1884. Annales de l'Institut Pasteur, 1887.

[4] Marchiafava and Celli: Fortschritte der Med., 1883, No. 18; 1885, No. 11; 1885, No. 24. Arch. per le Sc. Med., 1886, x., No. 9.

[5] Councilman: Medical News, January 15, 1887. Transactions of the Association of American Physicians, vol. i. Councilman and Abbott: American Journal of the Medical Sciences, April, 1885.

Dr. Mendelson asked whether any relationship possibly existed between the Hæmatozoön malariæ and the Filaria sanguinis hominis, since they had some points in common, namely, that both were blood-parasites; both appeared intermittently, giving rise to paroxysmal seizures.

Dr. James did not know of any connection between the two. Zoölogically they were quite distinct—the filaria belonging to the nematodes, the hæmatozoön to the protozoa. The presence of filaria in the blood was interesting, however, in that it suggested the possibility of a parent-organism existing within the body, and there discharging its young. It has been surmised that in malaria the parent-organism resides in the spleen, and there discharges its young, this discharge being the exciting cause of the malarial paroxysm. In the blood taken from a patient after the chill we find spores and pigmented amœboid forms, but whether these are a brood which will later

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[6] Osler: British Medical Journal, March 12, 1887.

[7] Leuckart: The Parasites of Man.

[8] Golgi: Arch. per le Sc. Med., 1886, No. 4. Fortschritte d. Med., 1886, p. 693. Gazzetta degli Ospitali, 1886, No. 72.

[9] Marchiafava and Celli: Arch. per le Sc. Med., X., No. 9, 1886.

[10] Gerhardt: Zeitschrift f. klin. Med., 1884, vii.

[11] Marchiafava and Celli: Fortschritte d. Med., 1883, No. 18; 1885, No. 11.

[12] Mariotti e Ciarrocchi: Lo Sperimentale, December, 1884.

[13] Zopf: Die Pilzthiere oder Schleimpilze. Breslau, 1885.

[14] Mosso. Klebs: Cent. f. Bact. und Parasit., Band ii., No. 1. Also, Klebs: Real Encyklopädie, Article Flagellata.

[15] Maragliano: Ueber die Resistenz der rothen Blutkörperchen. Berl. klin. Woch., 1887, No. 43.

[16] Pfeiffer: Zeitschrift f. Hygiene, 1887.

[17] Kartulis: Zur Aetiologie der Dysenterie in Aegypten, Virchow's Archiv, vol. cv. Zur Aetiologie der Leberabscesse, Centblt. f. Bact. und Parasitkde, ii., 25.

[18] Deichler: Deutsche Med. Zeitung, 1886, vii., 74.

[19] Pfeiffer: Monatsshefte f. Prakt. Dermatologie, 1887, No. 10; 1887, No. 13. Also, Loeff: Ibid., 1887, No. 5.

[20] Gaulé: Arch. f. Phys., 1880, p. 57. Ueber Wurmchen welche aus den Froschblut-Korpetchen auswandern. Also, Archiv f. Phys., 1881, p. 297. Gaulé: Cytozoen und Zellen, Biol. Cent., 1886, p. 345. Also, Centblt. f. d. Med. Wiss., 1881, No. 31.

[21] Lankester: Quart. Mic. Jour., 1882, p. 53.

[22] Wittich: Spirillen in Blut von Hamstern, Cent. f. d. Med. Wiss., 1881, No. 4.

[23] Mitrophanow: Biolog. Centralblatt, 1883, p. 35.

[24] Danielewsky: Die Hematozoen der Kaltblütter, Arch. f. Mik. Anat., vol. xxiv., p. 588. Cent. f. Med. Wiss., 1886, No. 41, p. 737.

[25] Lewis: Flagellated Organisms in the Blood of Healthy Rats, Quarterly Journal of the Microscopical Society, 1879, p. 109; 1884, p. 357.

[26] Crookshank: Journal of the Royal Microscopical Society, November 10, 1886. Also, Crookshank: Manual of Bact.

[27] De Bary: Morphologie und Biologie der Pilze, Mycetozoen und Baeterien. Leipzig, 1884.

[28] Zopf: Die Pilzthiere oder Schleimpilze. Breslau, 1885. Also, Zur Morphologie und Biologie der niederen Pilzthiere. Leipzig, 1885.

[29] Blanchard: Hématozoaires, Dictionnaire encyclopédique des Sciences médicales.

become mature we are as yet unable to say. Laveran regarded the flagella as the fully developed organisms.

Dr. Prudden wished to call attention to one point suggested by the paper. This was the change which is taking place in regard to the proof deemed necessary to establish an etiological relation between micro-organisms and disease. In the early period of modern bacteriological investigations—that is, those carried on by means of the methods devised by Koch—it was deemed necessary, in order to definitely establish an etiological relationship between a given species of bacteria and a disease in man, first, to show its constant occurrence in connection with the lesions of the disease; second, to completely isolate it by culture; third, to produce, by inoculation with the pure culture in some of the lower animals, lesions identical or closely analogous with those found in man; and, fourth, to demonstrate the presence of the micro-organism in the lesions. It is noteworthy that the first diseases completely investigated in the light of the new methods were those which are frequently found in both men and lower animals. But we have learned that there are other diseases, common in man, to which the lower animals are not only not naturally subject, but to which they are quite invulnerable by any of the experimental methods yet known to science. So that at present, while in every case positive results from inoculations of pure cultures are ardently sought after, we must always be prepared to find that one step in the chain of evidence must perforce be dispensed with. Unfortunate as this is, we should not forget that, with every acute infectious disease whose relationship to bacteria has been completely demonstrated, the morphological evidence alone becomes stronger in those other diseases which we cannot reproduce in animals.

Thus it was that the accumulation of morphological data such as Dr. James had laid before the Society in his paper assumed a significance greater than was evident before the limits of experimentation had become as well defined as they are to-day.

The President announced as the

COMMITTEE ON MICROSCOPY

for the ensuing year, Drs. J. S. Thacher, Ira Van Gieson, and H. M. Biggs.

The Society then went into executive session.

Stated Meeting, February 8, 1888.

W. P. Northrup, M.D., President, in the Chair.

The President presented, on behalf of a candidate, specimens removed from the body of a child that had died of *tubercular meningitis with general tuberculosis.*

Dr. G. R. Lockwood presented, on behalf of a candidate, a specimen of *hour-glass uterus.*

Dr. F. S. Sellew presented the history of a case of

CHRONIC BRIGHT'S DISEASE WITHOUT ALBUMINURIA.

The patient was a woman, aged sixty-two, unmarried, temperate; had been failing in health for five or six years, "but no doctor was able to tell her what was the matter." Two years ago she came under observation. At this time she was in fair flesh, very anæmic. No stomach symptoms, no headache. She complained of nothing except weakness in the knees. She could not walk as far as formerly, and yet went about out of doors. She said that her urine had been examined by six doctors, and that no one had found anything wrong with it. Repeated examinations made by Dr. Sellew gave the following results: Amount passed in the twenty-four hours, twenty-four ounces; specific gravity, 1.015, acid, pale, or amber-colored; no albumin, no sediment; no casts were ever found. During the last week of life a trace of albumin appeared.

There were no symptoms up to within three weeks prior to her death, save a gradually increasing feeling of weariness and exhaustion, together with increasing pallor. The pulse was regular and of normal tension until the last three weeks of her life, when there were occasional attacks of palpitation.

At this time there was diarrhœa, which alternated with œdema of the feet and ankles, but there was no ascites. There was occasional vomiting and moderate headache, but the principal symptoms of which the patient complained were excessive weakness and weariness. The pallor gradually increased and became waxy in nature.

The diagnosis of chronic Bright's disease was made at the outset, on the characteristic waxy pallor and the low specific gravity of the urine, and was later confirmed by the occurrence of vomiting and the œdema of feet and ankles, alternating with diarrhœa.

The autopsy, made twelve hours after death, by Dr. W. P. Northrup, showed the following: The heart was pale

and flabby, and contained decolorized clots; the muscle substance was so softened that the finger with moderate pressure could be easily thrust through both walls of the cavities together. The liver was slightly diminished in size, the surface of a chocolate color, finely nodulated and firm. The kidneys were small, the surfaces lobulated and finely granular, the capsules bringing away bits of tissue when stripped off; the cortex was of normal thickness, pale and firm.

The microscopical examination showed the heart muscle to be the seat of advanced fatty degeneration.

In the kidneys there was diffuse interstitial thickening with atrophy of the tufts, contraction of some of the tubules, and dilatation of others.

The epithelium was broken down and had disappeared in some of the tubules, and there were hyaline casts in many. These might have appeared in the urine from time to time, and easily have escaped discovery.

The anatomical diagnosis was made of: 1, Chronic diffuse nephritis; 2, advanced fatty degeneration of the heart; 3, cirrhosis of liver.

The points of special interest in the case were thought to reside in: 1, The absence of anything abnormal in the urine upon repeated examination of several doctors; 2, the characteristic waxy pallor, and, 3, the well-marked lesions shown by the autopsy.

Dr. Sellew also presented specimens, removed from the body of a child who had died from

PLEURO-PNEUMONIA, COMPLICATED WITH PLEURISY WITH EFFUSION OF THE OPPOSITE SIDE.

Fidelis M——, aged nine months, was brought to the out-door department of the New York Foundling Asylum on January 30, 1888, suffering from diarrhœa and vomiting, and with inflammation of both tonsils.

The child was wet-nursed and was fairly well nourished. On February 1st, the diarrhœa and vomiting were relieved, but the tonsils were still greatly swollen, though there was no membrane. On February 3d the tonsillitis had begun to subside, but there was a short cough, with labored respiration, and on the right side dulness and bronchial breathing were present. The dulness was not marked, nor did it extend over a large area of lung. On the left side there were a few coarse mucous râles.

On February 8th the child was returned to the Asylum in collapse, and died soon after.

At the autopsy it was found that the left lung was compressed by pleuritic effusion, there being three ounces of turbid serum in the pleural cavity. Upon the surface of the compressed portion of the lung there was a thick

exudate of pus and fibrin. The only aërated portion of the lung was the anterior half. The right lung was the seat of an extensive and severe pleuro-pneumonia, the entire lung being in a condition of red hepatization, without any aërated portions. The other organs, even the heart, were normal.

The interesting features of the case were the two associated conditions found in the two lungs. In one there was pleurisy, with effusion of serum and fibrin, with compression of the lung, but no pneumonia. In the other side was an extensive pleuro-pneumonia, the only aërated portion of the lungs being the anterior half of the left or compressed lung. It was thought that the disease commenced on the right side, and was complicated later on by the pleurisy with effusion.

It was also interesting to note the absence of pericarditis. It is very common in autopsies at the New York Foundling Asylum, in extensive inflammation of both lungs or in extensive pleural fibrinous exudates, to find associated pericarditis with excess of fibrin.

The President remarked upon the two especially interesting points in connection with this case. The first was the extremely severe nature of the lesions. The pleurisy with effusion compressing the lung on the left side, and the pneumonia with pleurisy on the right side, rendered both lungs almost useless, and for the last few days of life the breathing space was limited to a small portion of the right lung. The second point of interest was that the pericardium was entirely free from disease, although the pleural surfaces on either side of it were inflamed.

Dr. J. Lewis Smith said that it was not uncommon to find the pericardium involved under these circumstances, yet such was not the invariable rule. He thought it would have been interesting to study the physical signs present in this case. He also inquired whether there were any tubercles present.

The President said that unfortunately there was no clinical history of the case. He did not know which side was first affected, but for the few days immediately preceding death there would probably have been little in the way of physical signs except dulness on both sides. No evidences of tuberculosis had been found at the autopsy.

Dr. Mary A. Dixon Jones presented the

TUBES AND OVARIES

removed by operation from a woman, twenty years of age, married three years, no children. She suffered not only at the menstrual period, but in the intervals as well, with distress and pain in the pelvis. Laparotomy was performed, and the tubes and ovaries were found bound down

by adhesions. The tubes were enlarged, closed at each end, and apparently filled with pus.

Dr. Jones also presented another specimen, removed from a woman thirty-eight years of age, the mother of four children, the oldest ten, the youngest five years of age. The patient suffered much pain, and was not able to attend to her ordinary household duties. The cervix was found, on examination, to be deeply lacerated and widely gaping, as if the uterus were pulled in some unusual position. Apparently all around the cervix was a solid mass, having a radius of three and a half to four inches, and extending down on either side to the anal region, and upward as high as the top of the pubic bone. In some portions this tumor was so solid that it suggested a fibroid, but other portions were soft and even fluctuating.

THE UTERUS WAS PULLED OVER TO THE RIGHT,

and firmly fixed, seeming to be closely adherent to the mass about it. The sound could be passed up a distance of four inches.

When the abdominal cavity was opened the uterus was found fixed and deflected toward the right, the right appendages being below it and deeply embedded in adhesions. To the left of the uterus there was a solid mass occupying the whole left of the pelvis, and closely fixed and bound down by adhesions. That portion of the appendages originally anterior was turned over and adherent to the back of the pelvis, and was covered by the remains of strong and well-organized adhesions, which had to be separated with the greatest care on account of some being adherent to the rectum. The greatly enlarged tube, which occupied the lower part of the mass, after going a straight course of three inches, bent upon itself at a right angle and extended upward for another three inches, measuring altogether five and a half to six and a half inches in length. The lumen of the fimbriated extremity measured an inch and a half in diameter, being large enough for the entrance of the index-finger. By this turning over the posterior portion of the appendages had become the anterior, and, being closely united to the anterior wall of the pelvis, had thus formed a cavity, and it was here that the sense of fluctuation had been obtained. There were several other large cysts similarly formed by inflammatory adhesions. The whole of the mass was removed and the abdominal cavity was washed out, but the pedicle was so large and so fibrous that even after tying it still bled, so it was fixed in the abdominal wound by Koeberle's clamp.

The organs had not yet been examined microscopically, but the nature of the changes present might be inferred from those found in a case having similar general features

upon which Dr. Jones had operated. The patient was twenty-one years of age, married, and had two children. She menstruated first at the age of eleven years, and then, and ever since, had had severe pain, showing that she had begun her menstrual life with diseased ovaries. Since the birth of her last child the menstrual pain had increased in severity. The cervix and perineum were both deeply lacerated, and on account of the youth of the patient it was determined to repair these structures first, in the hope that the condition of the woman might be thereby so far improved as to render her life endurable without taking from her the possibility of future child-bearing. The operation was, however, not successful in producing this result, so the removal of the ovaries was decided upon. The appendages, which were buried in the midst of adhesions and extensively diseased, were also removed. The tube was enormously enlarged, especially at the fimbriated extremity, and completely closed. There were extensive adhesions back of the tube, and between it and the ovary, and numerous cysts had been formed in this new inflammatory tissue. The ovaries were small and buried in adhesions. Microscopical examination showed degeneration of the ovary from chronic ovaritis, the entire structure being changed except a small portion at the periphery. The tube was diseased in all of its structures—mucosa, connective tissue, muscular tissue, and peritoneal investment.

In many places the perimysium was almost wholly destroyed, and the smooth muscle-fibres of the tubes were entirely disintegrated by the inflammatory exudate. In many places the leucocytes were so crowded together as to form true abscesses, no vestige of muscular tissue being left. In other places, under the microscope, the muscle fibres presented the appearance of a few broken fragments floating in a sea of inflammation-corpuscles.

Dr. Jones objected to the term

INTERSTITIAL SALPINGITIS,

usually applied to this condition, as it was not the interstitial connective tissue that was affected, but the muscle itself. She thought the term "corporal salpingitis" would be more appropriate.

Salpingitis usually begins in the mucous lining of the tube and travels outward, but in this case the periphery was most affected, evidently by continuity from the peritoneal inflammation, while the mucosa was least involved.

It was perhaps of interest to add that the patient had at times suffered from great mental depression, and had often threatened self-destruction, but since the operation these symptoms had disappeared, and she had gained steadily in health and strength. Dr. Jones believed that in very

many cases similar to this, of supposed uterine disease, the most serious trouble was really with the ovaries or tubes.

Dr. J. Lewis Smith presented a specimen of

UMBILICAL PHLEGMON,

removed from a child who had died in the New York Infant Asylum. Diphtheria had appeared in this institution in August last, since which time there had been in the neighborhood of thirty cases of the disease, and among them were four in the new-born. During the same period there had been six cases of umbilical phlegmon, a most unusual number for that institution.

The patient, Violet M——, was born on January 5th. There was nothing unusual about the labor, and the mother has done well—indeed in all these cases of umbilical phlegmon the mothers had done well. The navel was dressed with borated cotton, according to the routine habit in the Asylum. On January 11th the baby was noticed to have a slight discharge from the umbilicus. Its temperature at this time was 100°. The following day there was some redness immediately about the umbilicus, and this extended gradually until, on the day succeeding, it covered an area with a radius of an inch and a half around the navel. On January 14th examination of the fauces revealed the presence of

DIPHTHERIA.

The temperature ranged from 98.3° to 101.2°, the pulse was weak and varied between 120 and 146. The throat was sprayed with a mixture of sodium benzoate and sodium bicarbonate, of each ʒ ij.; Listerine, ʒ j.; glycerine, ʒ ij.; and lime-water, ʒ viij. The exudate did not disappear under this treatment, however, and on the following day—when the patient died—it had extended to the nostrils.

Post-mortem examination.—The diphtheritic exudate was found covering the epiglottis, especially its under surface, and extending into the larynx down to a point just below the vocal cords. The lungs were mottled with hemorrhagic points. The umbilicus had been sent to Dr. Prudden for examination.

Dr. Smith regarded the occurrence of such an unusual number of cases of umbilical phlegmon during the local epidemic of diphtheria as a point of considerable interest, and he thought it would be of great value to determine whether there were any causal relation between the two.

As a matter of prophylactic interest, he said, it was worthy of mention that there had been no more cases of diphtheria since use had been made of turpentine steam

in the institution. A mixture was prepared of oil of eucalyptus ʒ ij. to iv.; turpentine, ℥ viij.; and water, Oij. This was kept in a constant state of ebullition night and day. If this were done everywhere he believed that there would be no danger of the physician carrying away the infection in his clothes.

Dr. Prudden reported as follows upon the specimen: A section through the abdominal wall parallel with the abdominal vein showed that the interstices of the abdominal parietes about the vein were densely infiltrated with serum, fibrin, and pus, and that a small red clot was firmly lodged in the outermost portion of the vein.

SECTIONS STAINED FOR BACTERIA

showed large collections of micrococci at one side of the clot in the vein, with a necrosis of the inner layers of the vein near the bacteria.

Furthermore, the exudation mass in the tissues outside of the vein, particularly along the adventitia, was intermingled with large numbers of micrococci, either scattered singly or lying in clusters or in larger and shorter chains.

Cultures made from the clot and from the exudation showed that two species of micrococci were present, which were readily identified as the *streptococcus pyogenes* and the *staphylococcus pyogenes aureus*.

As to the relationship between these bacteria and the diphtheritic process which developed later in the throat, Dr. Prudden could only say that, as we do not yet know what bacteria cause diphtheria, a definite opinion must be held in abeyance.

Dr. J. S. Thacher presented a specimen of

LARGE TUBERCULAR MASSES IN THE PERITONEAL CAVITY,

which had been sent to him by Dr. Montague L. Boyd, of Savannah, Ga., with the following history:

The patient was a negro girl, five years of age. The mother stated that the child had complained of pain in the abdomen two years ago, and that she had noticed a lump there which was tender on pressure. The case had been under observation for six months. The abdomen had been very much enlarged, the rest of the body being extremely emaciated. The bowels were regular, and the appetite fair. The temperature was almost constantly elevated, ranging from 101° to 103°. There was no œdema of the face or limbs, and no cough at any time. The abdomen became more and more enlarged, while the rest of the body gradually wasted away, the strength failed by degrees, and the child died without any other symptoms.

Dr. Boyd's diagnosis of the case was sarcoma, while

other physicians who had seen the child suggested malarial enlargement of the spleen and ovarian tumor.

The autopsy was made under adverse circumstances, and only the abdomen was opened. The peritoneal cavity contained about two gallons of a thin milky fluid. The tumor, with intestines, stomach, and spleen, was removed. The tumor was seen, on inspection, to be composed of a number of rounded nodules, some fused together and some separate from the rest. The tumor measured about $5 \times 3\frac{1}{2} \times 3$ inches, the largest distinct mass being about $4 \times 2\frac{1}{2} \times 2$ inches, and the others of all sizes less than this. They lay behind the peritoneum, in the mesentery, and in the omentum, and gave the impression of being enlarged lymphatic glands. On section they were found to be of a firm, cheesy consistence, and of a pale yellow color.

Under the microscope they were seen to be made up, for the most part, of a structureless granular mass, but at the circumference of inflamed connective tissue, having the features of tubercular inflammation fairly well marked. A very few tubercle bacilli were also found in the sections.

The Society then went into executive session.

Stated Meeting, February 22, 1888.

W. P. Northrup, M.D., President, in the Chair.

The President presented, on behalf of a candidate, a *kidney of a dog containing a large calculus.*

Dr. Frank Ferguson presented, on behalf of a candidate, a specimen of *cancer of the pylorus with secondary neoplasms in other organs.*

Dr. Ferguson then presented a specimen of

GUMMATOUS TUMORS OF THE LIVER,

the larger ones being about three-fourths of an inch in diameter. The patient was a man, about twenty-nine years of age, who was admitted to the Chambers Street Hospital a few hours before death. He was in a comatose condition on admission, and no history of the case could be obtained.

A second specimen was one of

ENCHONDROMA OF THE STERNUM,

removed from the body of a man who had died of general peritonitis following perityphlitis. The tumor was composed of cartilage with strands of fibrous tissue running through it, and in places small fragments of bone were found. It measured 12 ctm. in length, 7 in width, and $4\frac{1}{2}$ antero-posteriorly.

Dr. Mary A. Dixon Jones presented a specimen of a large

FIBROID TUMOR OF THE UTERUS,

removed by operation from a woman six days before. The patient was forty years of age, had been married twice, but never had had any children. Examination showed the presence of a large, solid, fibroid tumor of the uterus, filling the whole abdomen as far as the ensiform cartilage. The growth had evidently existed for a very long time, but had troubled her more especially during the last five or six years. The abdomen was opened, the tumor freed partly from adhesions, and a portion cut away. Then the clamp was secured lower down and another portion was removed. The pedicle thus secured was seen to consist of a mass of tumors, so it was decided to perform vaginal hysterectomy to remove the rest of the tumor.

The entire tumor-mass weighed thirteen and one-half pounds. The upper third consisted of a single solid mass, the remainder being composed of a number of smaller tumors, varying from one and a half to four and a half inches in diameter. The left ovary was enlarged, con-

taining a hæmatoma two inches in diameter. The tube measured five inches in length and was filled with pus, the fimbriated extremity being closed and adherent to the ovary. The right tube was enormously enlarged, and was coiled upon itself. The end, which presented no trace of fimbriæ, was closed, the tube measuring here three and four-fifths inches in circumference, and being filled with a bloody fluid.

Dr. D. Bryson Delavan related a case of

EPITHELIOMA LARYNGIS.

"A. S. J——; United States; aged fifty-eight; married; merchant. Family history excellent. Ancestors remarkably healthy and long-lived. Was brought up on a farm. Has always enjoyed perfect health. No diathesis discoverable. Has never used tobacco or alcohol in any form. Has lived much in the open air. In the summer of 1885 he caught cold, which affected his larynx. The resulting hoarseness never entirely disappeared, but became worse by degrees until the winter of 1886, when he became aphonic. About this time marked irritation of the larynx set in, causing considerable annoyance. Dysphagia followed, and later, dyspnœa. The latter became so severe that in June, 1887, he applied for relief at the Throat Department of the New York Polyclinic, having been referred by Dr. M. Allen Starr. On examination, the patient appeared a large, splendidly developed, vigorous man. His heart, lungs, and abdominal viscera, so far as could be ascertained, were normal. He was aphonic and was suffering from urgent dyspnœa. Laryngoscopic examination revealed the following condition: The mucous membrane of the larynx was deeply congested; the left arytenoid prominence was tumefied, the swelling extending over the whole of that side of the larynx; the left false cord was greatly enlarged and projected so far beyond the median line as to cause almost entire occlusion of the glottic aperture; the left vocal band was partially destroyed by a deep ulcer, which had its origin, apparently, in the adjacent sacculus laryngis, and which extended throughout the middle third of the band and downward below the glottis. There was no cachexia apparent and no involvement of the neighboring cervical glands. The appearance of the ulcer, together with the history of the case, at once suggested malignant disease, and, by reason of the urgency of the dyspnœa, an immediate tracheotomy was advised. To this the patient objected. On his return home the dyspnœa increased and by the following afternoon became so severe that asphyxia was threatened, and I was hastily summoned. Tracheotomy was performed and a cannula was inserted, to the patient's immediate relief. Microscopical

examination of fragments of the tumor proved it to be epitheliomatous. Following the introduction of the tube, as is common in such cases, the condition, both general and local, improved, and all of the laryngeal lesions ameliorated to a marked degree. About this time he was seen in consultation by Dr. William T. Bull, who advised laryngectomy. This he would not permit until, as he said, he had seen the result of a similar case upon which Dr. Bull had just operated. The danger of delay was urged, but his decision was unalterable. During July the improvement ceased, and in August the disease again began to make steady progress. When, in the latter part of August, having seen the brilliant result attained in the case above mentioned, he at last consented to an operation, it was found that the whole of the thyroid, the cricoid cartilages, and even the hyoid bone, were involved, and the neighboring tissues were infiltrated, so that extirpation of the larynx was no longer justifiable.

"The progress of the disease was now rapid, the whole interior of the larynx becoming involved, and the cartilages increasing in size until, finally, they attained about four times their usual dimensions. There were profuse and annoying expectoration, cachexia, considerable emaciation and insomnia, but little dysphagia, and that easily relieved by the application of cocaine, a fair degree of general strength, and no glandular involvement.

"The patient's condition grew gradually worse until January 1, 1888, when he complained that the tube seemed to be obstructed, and that removal and cleansing of the inner cannula did not relieve it. Examination of the trachea with a soft, flexible probe revealed the presence of a mass of tissue, of soft, friable consistence, upon the left side of the trachea and extending downward below the end of the tube. The removal of the most prominent part of this obstruction gave relief to the dyspnœa for several days; the attendant was instructed to remove the tracheal tube and insert far down the trachea a large soft catheter whenever an attack of dyspnœa occurred. This succeeded in temporarily relieving the tracheal stenosis, but the attacks of dyspnœa came on with increasing frequency, and during one of unusual severity the patient died.

"The principal points of interest, pathologically, in the above were, first, the extension of the growth into the trachea to so great a distance; and second, the fact that the adjacent glandular structures remained uninvolved."

Dr. J. West Roosevelt presented

A SERIES OF HEART LESIONS.

I present the following eight specimens of lesions of the heart which illustrate certain pathological and clinical

facts worthy of note. In preparing these specimens I studied the records of eight hundred and fifty cases which came to autopsy at Roosevelt Hospital. The cases were taken as they came, and embrace various diseases and injuries. I looked for cases of valvular disease in which there was stenosis or insufficiency of a valve, or in which there were vegetations found on one. Cases of malignant endocarditis, and cases of valvular insufficiency from dilatation of the heart or ascending aorta, are excluded.

Among the 850 cases I find 123 of valvular heart disease, or 14.1 per cent. In these the mitral valve alone was diseased in 50; stenosis existed in 15; insufficiency in 27. Both stenosis and insufficiency are noted in 6; vegetations, not producing stenosis or insufficiency, in 2. The aortic valve alone was diseased in 24 cases. Stenosis existed in 2; insufficiency in 12; both, in 3; vegetations, not producing either, in 7. Both aortic and mitral valves were diseased in 44 cases. The following combinations were found: Aortic and mitral insufficiency in 13; mitral stenosis and aortic insufficiency in 8; aortic and mitral stenosis in 5; aortic stenosis and insufficiency, and mitral stenosis and insufficiency, in 3; aortic insufficiency and mitral stenosis and insufficiency in 3; aortic stenosis and insufficiency and mitral stenosis in 3; aortic stenosis, mitral stenosis and insufficiency in 1; vegetations on both valves, without interference with functions, in 2; vegetations on aortic valves with mitral stenosis, in 3; vegetations on aortic valves with mitral insufficiency, in 2; vegetations on mitral valve with aortic stenosis, in 1. The tricuspid alone was insufficient in one case. It alone was covered with vegetations in one case. In one case there were vegetations on the tricuspid, mitral, and aortic valves, the mitral being stenosed; and in one there was tricuspid insufficiency with aortic and mitral stenosis and insufficiency.

In these cases the most common lesions are those of the mitral valve. The next most common are combinations of aortic and mitral lesions. Then we find lesions of the aortic valves, and lastly, those in which the tricuspid, alone or in combination with other valves, is diseased. I would call attention to the rarity of cases of pure aortic stenosis, two only being found.

In the series the males exceed the females in the proportion of 94 to 29. The excess is due, partly at least, to the fact that many more males than females come to the hospital, and partly to the fact that in a number of cases death was caused by injuries to which the male sex is more exposed. A distinct history of acute articular rheumatism is found in 44, among whom 10 were drunkards and 1 had syphilis; 3 gave a history of both rheumatism and Bright's disease. Chronic diffuse ne-

phritis seemed to precede the cardiac lesion in 20 cases, and in 5 of these there was a history of intemperance. A clear history of syphilis was noted in 6 cases, two being drunkards; none had had rheumatism. Alcoholism alone is mentioned in 7 cases. No mention of any of these causes occurs in 24 cases, as the histories are imperfect. In 7 all of them are denied; in 2, scarlatina and measles are noted. Four cases of sudden death are recorded, 3 with mitral insufficiency, one aortic insufficiency. In none of these cases was death sudden, in the sense of having been preceded by no symptoms, but the end came suddenly, the patients having done well before. Death occurred from accident or from disease which was not dependent upon the heart or kidney lesions in 22 cases. Uræmic coma or convulsions terminated 8; 3 died of cerebral hemorrhage, 3 of acute pericarditis, and the remainder died with the usual symptoms of dropsy, dyspnœa, etc.

In attempting to draw any conclusions from these cases, the following points should be borne in mind: First, as to the relative proportion of cases of valvular disease to other diseases in hospital cases in New York, the number of autopsies studied is, I think, sufficient to show about the right proportion, namely, about fourteen per cent. Second, as to the one hundred and twenty-three cases noted, no reliable deductions can be drawn, as they are not numerous enough. Third, the excess of males over females is probably too great, for the reasons mentioned. Fourth, in studying the etiology it must be remembered that in twenty-four cases the history is imperfect, and that these must be excluded. Fifth, that cases of sudden death do not often come under observation in a hospital, and are more frequent than the figures show. Sixth, cases dependent upon acute infectious disease are not fairly represented, because few children are treated in Roosevelt.

Bearing these facts in mind, we find that rheumatism was present in nearly forty-four per cent., chronic diffuse nephritis in nearly twenty per cent., syphilis in nearly six per cent., alcoholism alone in nearly seven per cent.; but that, taking cases with rheumatism, chronic diffuse nephritis, and syphilis, as well as cases without these, alcoholic excess was noted in nearly twenty-four per cent.

Aortic Insufficiency.—Calcareous Plates in Aorta Surrounding the Origin of the Coronary Arteries —Angina Pectoris.—Male, aged forty-six; admitted May 28, 1886. No rheumatism. Syphilis twenty-five years ago. Alcoholic habits marked at one time. No cardiac, pulmonary, or renal symptoms, except the attacks of angina. Attacks first began five weeks ago. First, has

oppression in chest and dyspnœa. Then severe pain in *right* side of chest, radiating over whole front of chest; felt in both shoulders and both arms. During attack, fears immediate death. Shortly after admission, had an attack. The pain was severe. The radial arteries were hard and cord-like. Sweating profuse. Under amyl nitrite and magendie pain subsided for an hour. Then was seized with abdominal cramps without chest pain. Subsided under magendie. At 3 A.M., May 29th, after going to closet, patient returned to bed and suddenly died.

Autopsy May 29th, at 1 P.M., by Dr. Delafield. Rigor mortis very marked. Well-nourished man. All organs normal but congested, except the heart. The left ventricle is slightly dilated and thinned. The heart is not contracted, and the cavities contain a moderate amount of red clotted blood. The aortic valves are thickened and insufficient. A good deal of degeneration of the aorta. Just above the aortic valves the changes are very pronounced, and there is a calcareous plate which surrounds the origin of the coronary arteries. The coronary arteries themselves are perfectly normal. The mitral valve is a little thickened.

This case is interesting, as showing the relation of interference with the coronary circulation to attacks of angina.

CHRONIC PERICARDITIS WITH ADHESIONS AND CALCIFICATION.—AORTIC AND MITRAL STENOSIS—HYPERTROPHY OF THE LEFT VENTRICLE—ABSENCE OF CHRONIC, PASSIVE CONGESTION—DEATH FROM SLIGHT CAUSES.—Christopher B——, aged fifty-six; German; married; conductor; admitted to Roosevelt Hospital at 2.05 A.M., January 15, 1888.

Had been drinking hard for five or six weeks. Between 1 and 2 A.M., to-day, attempted to commit suicide by cutting both arms in the fold of the elbow. Wounds, about three inches long, had not divided the arteries. He did not seem to have lost much blood. He was conscious. Pulse fair strength, but rather rapid. Respiration normal. Had several slight convulsions, and died at 6.50 A.M. Heart was not examined during life.

Autopsy January 15, 1888, at 2.30 P.M., by Dr. Delafield. Body well nourished. Thick layer of subcutaneous fat. The wounds in the arms have only divided the superficial veins and have not injured the arteries. The organs generally were not anæmic. There was no congestion, such as is usual with heart disease. The liver was fatty. The kidneys were normal, save that the surfaces were a little irregular, and the fat about the pelvis was increased. The stomach, intestines, and spleen were normal. There were some old adhesions over the lungs.

I show you the heart. The pericardium is firmly ad-

herent everywhere. In it, near the base, are some old calcific plates. Surrounding the tricuspid orifice, except that portion of it which is next to the mitral, is a ring of calcification which divides the auricle from the ventricle, and infiltrates the substance of the heart. The aortic valves are stenosed, and show numerous vegetations. The mitral valves are stenosed and thickened. Both of these valves show evidences of long-standing chronic endocarditis. The left ventricle is hypertrophied, but not dilated. There were post-mortem clots in all the chambers, and in the right ventricle some small ante-mortem clots.

The case is interesting, because it shows how little damage is done to the body in some cases by severe disease of the endocardium and pericardium. This heart must have been seriously crippled both by the valve lesions and the adherent pericardium, yet there was no passive congestion produced, nor did the man's nutrition suffer. Adhesions of the pericardium are not very uncommon. When such extensive lesions as those seen in this heart exist, one would expect much interference with the heart action, and marked symptoms and lesions of cardiac disease.

Another point of interest is that, although the man's heart seemed able to do its ordinary work, yet, after he had been on a prolonged debauch, a not severe hemorrhage was sufficient to kill him. He did not present the symptoms of severe hemorrhage. It is possible, of course, that, in addition to cutting his arms, he may have taken some alkaloidal poison, but he did not have symptoms of poisoning by any ordinary alkaloid, and it is most probable that his death was due to the exhausting effects of his spree and the slight hemorrhage, which were too much for the diseased heart.

Aortic Stenosis and Insufficiency.—General Dilatation of the Heart—Dilated Right Auricle Lying against the Chest Wall, at the Right of the Sternum, Causing Pulsation and Thrill, Simulating Aneurism.—Thomas K——, aged sixteen, admitted to Roosevelt Hospital August 3, 1881. Family history unimportant; denies syphilis, but has indulged in sexual intercourse; is temperate.

Rheumatism three years before admission. At that time had præcordial and epigastric pain, and has suffered from attacks of similar pain from time to time ever since. For one year has had frequent palpitation of heart and pretty constant dyspnœa, worse on exertion. Never had œdema of face or feet. Noticed no trouble with the urine. He has had frequent passing attacks of coughing, but during the past three months has suffered from constant cough, worse in the morning and evening, with mucopurulent expectoration. The cough is very violent, and

sometimes a paroxysm produces vomiting. Never spat blood. For the last month has sweat much at night; has been losing flesh and strength.

On admission patient is cyanozed. Urine acid; specific gravity, 1.026; without albumen. Has marked dyspnœa and orthopnœa.

Physical examination: Marked visible pulsation of the superficial arteries; typical "water hammer" pulse; heart apex beat in sixth space, one inch to the left of left mammary line; impulse diffuse and heaving; area of dulness increased. In the first and second spaces to the right of the sternum is an area of dulness, about two inches in width, beginning at the right border of the bone. Over this area is felt a systolic thrill. Auscultation over the same area gives a loud, rasping, systolic, and softer blowing diastolic murmur. A systolic murmur is audible in carotids and subclavians on both sides, about equally clearly. At the level of the third right costal cartilage the quality of the systolic murmur is entirely different from that heard above and in the vessels. It is very sharp and whistling. The diastolic murmur also, at this point, becomes loud and harsh; it is transmitted down the sternum to the ensiform cartilage. A short, blowing, systolic murmur is heard at the apex and to the left of it. Systolic murmurs can be heard in both vertebral grooves. A double murmur is very distinctly audible in the left supraspinous fossa. Markedly exaggerated breathing and voice at the right apex. Over the rest of both lungs subcrepitant râles and diminished breathing.

The patient spat considerable blood a few days after admission. His dyspnœa improved.

During his stay in the hospital the patient had several attacks of severe dyspnœa, accompanied by severe pain in præcordium and down the left arm, and marked sweating. He also suffered at times from pain in the epigastrium, which radiated through to the back. After being in the hospital about seven months he was greatly improved. His attacks of pain became less frequent, the cyanosis disappeared, and on April 6, 1882, he was discharged improved.

I wish to direct attention to the fact that there was an area of dulness at the right of the sternum, in the first and second space, and that over this area a systolic thrill could be felt and a double murmur heard, and that there was well-marked "water-hammer" pulse. It is worthy of note that the quality of the double murmur changed at the level of the third right costal cartilage. It is possible that a marked increase in intensity may have been mistaken for a change in quality, but I think this is not so. A diagnosis of aneurism of the ascending arch, with aortic

stenosis and insufficiency, was made at the time of his discharge.

The patient was readmitted June 21, 1884. After leaving the hospital he remained in pretty good health, suffering apparently, at times, a little from præcordial and epigastric pain. He was able to work, however. He had some cough. Two weeks before readmission, after exposure to cold and damp in an ice-house, cough became worse, and he suffered from cramps in the abdomen and legs. He also had pain in the right side of his chest; he lost appetite; sweats a good deal.

On admission he suffers from pronounced orthopnœa; has severe dyspnœa on exertion; urine alkaline; specific gravity, 1.030; albumen abundant; epithelial and hyaline casts.

At this time there are numerous moist râles heard over the bases of both lungs. Heart apex beat in same position as before. Diffuse and heaving impulse. Cardiac dulness enlarged. In the first, second, and third interspaces, for a distance of about one inch to the right of the sternum, there is dulness, and distinct pulsation with systolic thrill. Harsh systolic murmur can now be heard over the whole chest. Diastolic murmur audible the whole length of the sternum. Visible pulsation of carotids and subclavians, and over these vessels a systolic thrill. Finger in supra-sternal notch is lifted with the systole. Water-hammer pulse. Præcordial bulging.

Patient suffered from dyspnœa pretty constantly. He had a number of angenoid attacks. Profuse sweating was a prominent symptom. Orthopnœa was present until the end. On June 27th, at 4.10 A.M., became cyanotic. At 4.20 died.

Autopsy made by the writer eight hours after death. Body poorly nourished. Rigor mortis fairly marked. Brain not examined. The kidneys, spleen, intestines, and stomach were in a condition of passive congestion. The liver was firm and congested, and the upper surface showed a depression where the enlarged heart had rested upon it. The left lung was in a condition of pigment induration. The right pleural surfaces were everywhere adherent. The lung was in a condition of pigment induration. The lower lobe was compressed by the enlarged right heart. The bronchi were congested in both lungs, and contained muco-pus.

The heart weighed two pounds when emptied of clots. All of the chambers were dilated; their walls were about as thick as those of the normal heart. The aortic valves were stenosed and insufficient. One cusp is almost gone, and the others are thickened. Mitral valve thickened, but seems sufficient. Tricuspid and pulmonary valves

normal. The dilated right auricle lay against the chest-wall behind the second and third ribs to the right of the sternum, and it was this which had given the dulness, thrill, and pulsation described. The aorta was healthy and not dilated.

The case was peculiar because of the close resemblance to aneurism in the physical signs. When the patient returned, after an absence of two years from the hospital, it was thought that if he had had an aneurism it would probably have killed him before that time. The absence of symptoms of pressure upon the trachea or bronchi, œsophagus, or recurrent laryngeal nerves, and the absence of pain in the neighborhood of the pulsating area at the right of the sternum, together with the youth of the patient, were all against aneurism. On the other hand, the dulness, thrill, and pulsation on the right of the sternum were pretty strong evidence in favor of that diagnosis.

Acute Pericarditis with Effusion Complicating Double Lobar Pneumonia.—Male, aged nineteen; laborer; Austria; admitted February 13, 1888. History imperfect, because patient could speak no English. Sick eight days. Pain in right chest. No chill. When admitted was very weak. Restless. Pulse weak—94. Respiration, 34. Temperature, 102.8°. Extreme dyspnœa. Urine, specific gravity 1.019; trace albumen; no casts. The physical examination gave signs of consolidation over lower part of both lungs, behind. Over the right chest, in front, the resonance was normal. The breathing and voice were normal from the apex to the second rib, and thence to the bottom were broncho-vesicular. Over the left chest, in front, dulness began at the second rib and was found from thence down. Breathing broncho-vesicular. Heart apex in normal position. Sounds obscure, but no murmur. Patient sank and died February 15th, at 8.10 A.M. Autopsy showed red hepatization of lower lobes of both lungs, with a very thick layer of fibrin on the right side. The hepatized right lung had prevented the pericardial fluid from distending the sac on that side. The left lung was pushed backward and upward by the pericardial effusion. The pericardium was distended with purulent serum. I show the heart, which has no endocardial lesions. The visceral and parietal layers are covered with a pretty thick layer of recent fibrin.

The case is interesting because of the position of the effusion caused by the right lung, and also as illustrating a not infrequent, but often unrecognized, complication of lobar pneumonia. The absence of friction sound, I presume, was due to the effusion. Possibly, could the patient have been made to lean forward, it might have been heard.

ADHERENT PERICARDIUM.—This heart was taken from the body of a man who died in Roosevelt Hospital July 18, 1886, of meningeal hemorrhage. No history could be obtained, except of the fatal apoplectic attack. The autopsy showed meningeal hemorrhage and some phthisis. There were no evidences of chronic venous congestion. The visceral and parietal layers of the pericardium are everywhere firmly adherent. The walls of both ventricles are somewhat thinned. The valves are normal.

This lesion is not very rare. It is probably but rarely diagnosed during life, however. I looked through the records of the autopsies made at Roosevelt Hospital since 1880, and among these I find a number of cases. The lesion does not necessarily do the patient much harm, when uncomplicated by valvular disease, or at least it may exist for some time without producing the ordinary lesions of passive congestion. In this case there were no evidences of such congestion.

In another case, a young man of extraordinary muscular development worked as a roofer, and carried very heavy weights, and gave absolutely no history of anything pointing to heart disease. He died of disease of the ependyma with serous effusion into the ventricles of the brain. His pericardium was firmly adherent throughout; the heart, otherwise, was normal. There was no history of rheumatism or syphilis, and the time at which the adhesions were probably formed is unknown.

In looking for other cases I find only two in which the lesion was uncomplicated by valvular disease. In one, there was also chronic peritonitis and ascites, and the heart was normal, save for the adherent pericardium. The other was a case of tubercular pericarditis, which has been reported to this Society. In this case the heart-muscle was much thinned. In neither of these cases was a diagnosis made. As very little is really known about the physical signs produced by this lesion, I tried to see whether the records would throw any light upon them. In the case of the young man who died of disease of the ependyma, the heart is recorded as normal. In the case in which chronic peritonitis existed, it is simply stated that the heart action was irregular, intermittent, and feeble, and that there were no murmurs. In the case of tubercular disease, it is recorded that the heart impulse was imperceptible and the heart action weak. In another case of complete adhesions, which were very thick, there were no signs noted save the murmurs caused by complicating stenosis and insufficiency of the mitral valves. A fifth case gave the usual murmurs of aortic stenosis and insufficiency and mitral insufficiency which existed, and, in addition, it is noted that "there was visible pulsation with perceptible impulse in the third,

fourth, and fifth spaces at the left of the sternum." In a sixth case, with thickening and insufficiency of the aortic and mitral valves, the usual murmurs were heard and the impulse of the heart was felt in the sixth space, nipple line, and is noted as "forcible, but diffuse."

These, with one mentioned in another part of this paper, are the only cases I could find in which the physical signs are recorded. In none, so far as I could ascertain, was a diagnosis made of the pericardial lesion. It is probable that the possibility of its existence may have been forgotten, and that, therefore, some more distinctive signs were overlooked. It is very likely that, in all of the cases, the apex beat would have been found not to change its position when the patient's attitude was changed; and it is possible that the impress on the left of the sternum noted in the fifth case may have occurred with diastole, and not with systole. I shall not weary you by mentioning the physical signs usually described as indicative of this condition, but simply record the cases and call attention to the difficulties in diagnosis in adults.

Mitral Stenosis and Insufficiency.—Tricuspid Insufficiency—Dilatation of Both Auricles and of the Right Ventricle.—Patient, a boy aged fifteen, had scarlatina, followed by some pains in the joints, twelve years ago. Came under observation January 26, 1888. No history of cardiac symptoms until three weeks ago. Then cough, followed in a week by œdema of the feet and legs, abdominal swelling, and some dyspnœa. Grew rapidly worse. Dyspnœa and orthopnœa became extreme. Remained in hospital for about three weeks and was then taken home. Died February 15th. When admitted to hospital, urine was acid, specific gravity 1.027; small amount of albumen. He was very dropsical, and suffered much from dyspnœa and orthopnœa. The apex beat was in the fifth space, nearly in the left anterior axillary line. Impulse was diffuse and slapping, and could be seen causing indentations in the third, fourth, and fifth spaces to the left of the sternum. There seemed to be a drawing in of the lower part of the præcordium with the systole, but the heart action was too rapid to be sure of this. A distinct systolic thrill could be felt over the whole præcordium. Area of cardiac dulness enlarged. Systolic pulsation of veins of neck very marked. Rough, loud præsystolic murmur audible to the left of the sternum from the third rib to the apex. This murmur sounded almost like a friction sound. Systolic murmur over whole præcordium, loudest above the apex, transmitted to the left, and heard in the left vertebral groove. Pulmonary second sound much accentuated. The pulse was small but regular—110; respiration, 28; temperature, 101.8° F. There were signs of

fluid in both chests and abdomen. The apex-beat was hardly at all changed in position by such changes of posture as the patient could be made to take, but owing to his distress it was not possible to test this point thoroughly. From the signs I thought the boy had mitral stenosis and insufficiency, with dilated right heart; and from the apparent systolic depression thought he probably had an adherent pericardium. At the autopsy we were only permitted to examine the heart. There was ascites and hydrothorax. The pericardium contained a very slight excess of fluid, but was otherwise perfectly normal. The heart I present to you. The left ventricle is very slightly dilated. The aortic valves are very slightly thickened, and on one cusp is a small recent vegetation. The mitral valve and chordæ tendinæ are very thick. The cusps of the valve are glued together so as to make a rounded opening less than one-fourth inch in diameter, which evidently cannot be closed. The left auricle is dilated and thickened, its endocardium is thickened and white. The right ventricle is much dilated; the tricuspid valves are much thickened, their chordæ tendinæ are thickened and shortened. The right auricle is much dilated. The pulmonary valves are normal. As the heart lay in the chest the dilated right ventricle was in front of the left, and so great was the dilatation that the heart had been caused to rotate on its axis, so that the left ventricle was not only behind, but its left border was about one inch to the right of the right ventricle. The dilated right auricular appendix pressed upon the aorta, lying between it and the chest-wall.

Mitral Stenosis, Aortic Stenosis, and Insufficiency.—Tricuspid Insufficiency—General Dilatation of the Heart with Hypertrophy—Systolic Murmur most Distinct at the Apex.—The patient's history is of the ordinary symptoms of cardiac disease for one year. He was fifty-two years of age. He had had rheumatism, scarlatina, chancroids, and gonorrhœa. The only point of interest for my present purpose is that the only murmur audible was a systolic one, which could be heard over the whole præcordium, but was most distinct at the apex.

His heart is hypertrophied and dilated, and there is calcification, with stenosis and insufficiency of the aortic valves; very close stenosis of the mitral valve, and well-marked insufficiency of the tricuspid valve.

In connection with this case I show another heart with stenosis and insufficiency of the mitral valve, in which a systolic apex murmur alone was audible.

In the records of the Roosevelt Hospital I find the following cases, which are of interest when considering the relations of heart murmurs to lesions. It is needless to

say that they are all cases in which an autopsy was made:

1. Area of cardiac dulness enlarged. Feeble and irregular action. No murmurs. Lesion: Aortic stenosis and insufficiency. Mitral insufficiency. Hypertrophy with dilatation of left ventricle.

2. Heart enlarged. Feeble and irregular action; no murmur. Aortic valves thickened. Mitral valves stenosed. Hypertrophy of right ventricle.

3. Systolic apex murmur. Lesion: Mitral valve stenosed, and insufficient and calcified. Hypertrophy with dilatation of right ventricle. Slight dilatation of left ventricle without hypertrophy.

4. Systolic apex murmur, loudest at the lower end of the sternum. Lesion: Aortic insufficiency. Mitral stenosis and insufficiency.

5. Loud systolic murmur over the whole præcordium. Lesion: Aortic valves calcified, rigid, and insufficient. Mitral a little thickened. Great dilatation of left ventricle.

6. Systolic apex murmur. Lesion: Stenosis and thickening of the mitral valve. General hypertrophy and dilatation.

7. Systolic base murmur. Force of heart action great as compared with feeble pulse. Lesion: Mitral stenosis so marked as nearly to close the valve.

8. Forcible impulse against chest-wall. Irregular action. Heart-sounds obscure, but no murmur. Increased area of præcordial dulness. Small, weak pulse. Lesion: Hypertrophy of right ventricle. Thickening and stenosis of mitral valve. Marked thickening of the endocardium of the left auricle. Left ventricle normal.

9. Loud systolic murmur heard at apex, and transmitted to the left. Diastolic murmur over aortic valves, and transmitted down the sternum. Lesion: Dilatation and hypertrophy of the left ventricle. No valvular disease. This patient died of uræmia, due to atrophic chronic diffuse nephritis. It seems probable that the dilated heart permitted regurgitation through the aortic and mitral orifices during life.

10. Heart action forcible; rhythm somewhat irregular. A loud systolic, rough murmur heard over the whole præcordium, loudest at apex, and transmitted toward the axilla. No mention of possible transmission up great vessels. Lesions: Left ventricle normal. Right ventricle somewhat dilated. Pericardium distended with clear serum. Mitral and aortic valves are both much thickened and stenosed.

11. Double murmur, heard loudest at the apex and transmitted to the left. Lesion: Aortic valves covered

with old vegetations, ulcerated, and partly calcified. Old pericardial adhesions.

Of course, it is well known that murmurs do not correspond to lesions with absolute exactness, but it is interesting to see how little relation they may have to valvular disease. Among these cases are two with feeble and irregular action, but no murmurs, although the valves were diseased. The feebleness of action may have explained the absence of the murmurs. The murmur produced by mitral stenosis, if we may judge from these cases, is sometimes systolic and loudest at the apex, sometimes systolic and heard only at the base, and sometimes not audible. I do not speak of these cases as showing anything new, but I wish merely to emphasize something old and well known, but often forgotten. I do not wish to waste words, as some are now doing, by theorizing about the mode of production of murmurs; nor to worry about the possibility of the murmur of mitral stenosis being caused by systole of the auricle or the ventricle; nor whether it should be called "præsystolic," or "post-diastolic," or anything else. We have a murmur which precedes the first sound, and is usually only heard at or near the apex, and this indicates mitral stenosis. The murmur is of a generally peculiar quality, and begins softly and ends much louder. We have also in mitral stenosis a murmur of the same quality, which may take the place of the first sound; we may have no murmur, or the murmur in some cases seems actually to follow the sound. These facts are important. As for the theories, we do not know enough yet to really prove any of them. The Pathological Society is not a place in which we should discuss theories too much. The aim of these meetings is to give opportunities to study lesions and to learn from facts. From the study of lesions we can say: That extensive valvular disease may exist without murmur; that murmurs are not always heard in the situations usually described, nor are they always transmitted in the classical manner; that the murmur of mitral stenosis is peculiarly apt to be irregular in its time and situation; and finally, that although basic systolic murmurs, transmitted up the great vessels, are not uncommon, yet aortic stenosis is a rare lesion, and these murmurs usually only indicate disease of the valves without stenosis. I repeat that I present these facts not because they are new, but because they are so often forgotten.

In conclusion, let me summarize the facts which I have desired to illustrate by this series of hearts.

First, the specimen of calcareous plates in the aorta, obstructing to a considerable degree the coronary circulation. This specimen shows the influence of obstructed coronary vessels in producing angina pectoris.

Second, the case of pericardial adhesions and calcification with valvular disease. The point of greatest interest here is the death of the patient from a slight hemorrhage. It shows how feeble is the vitality in some cases of organic heart disease, even when the lesion of the heart has neither yet interfered with the nutrition of the patient nor produced passive congestion.

Third, the case of adherent pericardium is presented, with the epitomes of other cases, to call attention to the lesion, and also to the fact that its diagnosis is not easy.

Fourth, the acute pericarditis complicating pneumonia is presented to show the lesion, and to bring to mind a complication often overlooked.

Fifth, the case of valvular disease with general dilatation, and with dilated right auricle pulsating to the right of the sternum, is reported because of its resemblance to aneurism.

Sixth, the case of extreme mitral stenosis and insufficiency, with tricuspid insufficiency, is interesting because of the lesion and the physical signs.

The seventh and eighth cases, and the histories of certain others, illustrate the peculiar physical signs produced by some valvular lesions.

Dr. Frank Ferguson presented a specimen of

TUBERCULAR PERICARDITIS,

showing marked thickening of both layers of the pericardium. The patient was a boy, ten years of age, born in Italy. For a year he had been in the habit of drinking from three to seven glasses of beer a day, and had smoked cigarettes excessively. One year ago he had a violent cough, accompanied by fever, which lasted for ten days, during which time he was delirious. There was no expectoration, but the boy had pain in the right side of the chest and was unable to lie on that side. There was no dyspnœa. This attack lasted for two months, and left the patient weakened and drawn over to the left side. Two months ago he had a similar attack, and for a year he had had some fever and a cough in the morning. One week before admission to the hospital, on November 5, 1887, he began to have increased cough and fever. There was no chill. The patient had dyspnœa and pain in the right side on coughing. The fever lasted but one night, and there was no expectoration nor spitting of blood.

Physical examination.—The right side of the chest was markedly retracted, and expanded scarcely at all during respiration. There was dulness anteriorly at the apex. Vocal fremitus was exaggerated, and there were many fine moist râles. On the right side, posteriorly, below there was dulness—lost vocal fremitus, and diminished breath-

ing. The cardiac action was rapid—no murmur. The abdomen was distended, and the abdominal veins were markedly enlarged. There was a well-marked sensation of fluctuation. The liver extended from the fifth rib to a full hand's breadth below the free border, and it was somewhat tender.

Both chests were tapped and clear serum was withdrawn from the left. Urine yellow, acid, 1.006; no albumin. The patient became progressively worse, complaining of pain, sleeplessness, and dyspnœa, and getting weaker daily. He died on November 27th.

Autopsy.—Four hours after death. The peritoneum contained 1,500 c.c. of straw-colored serum. The diaphragm was on a level with the fifth interspace on each side.

There were firm old adhesions everywhere on the right side, and the pleura was much thickened. The pleural cavity contained lymph. On the left side were found old adhesions at the apex and 1,000 c.c. of clear serum. The upper lobe of the left lung was in the condition of gray hepatization. The lower lobe was œdematous. The right lung was firmly compressed and contained no air.

There were recent adhesions everywhere between the two layers of the pericardium. The heart was drawn toward the right. Its cavities were moderately dilated, but the valves were competent and the endocardium was normal.

Dr. H. M. Biggs presented a specimen of

ULCERATIVE ENDOCARDITIS.

The patient had been an attendant at the Charity Hospital for a number of years. He had never complained of any heart-trouble, and was sick only about eighteen hours before his death. On post-mortem examination both lower extremities were seen to be covered with a large number of petechiæ. The heart was hypertrophied and dilated, following an old pericarditis with obliteration of the pericardial sac. The mitral valve was the seat of an old trouble, as shown by the presence of some calcareous nodules, and also presented the lesions of recent endocarditis. The case was interesting on account of the sudden onset and short duration of the symptoms, and also because the changes in the heart were not of such a nature as to furnish the source for the numerous emboli necessary to account for the large number of petechiæ on the lower extremities. There had been a number of cases of small-pox in the hospital, and the man had had charge of the clothes of these patients. It was possible, therefore, that the case was one of hemorrhagic small-pox.

A second specimen was one of

RUPTURE OF THE HEART,

occurring in a man, fifty-eight years of age, a stone-cutter by occupation. He was apparently perfectly well, with the exception of some dyspnœa, on retiring one night, and was found dead in bed the following morning. At the autopsy the pericardium was found filled with blood, which had escaped through a small rupture in the antero-lateral wall of the left ventricle. There was no thinning and little, if any, dilatation of the heart. There was a clot along a branch of the coronary artery, but the vessel was almost perfectly normal, and was not ruptured.

Dr. Biggs also presented a third heart, removed from a patient in whom there had been heard, before death, a

MUSICAL SYSTOLIC APEX-MURMUR.

Death occurred from pulmonary trouble, and at the autopsy no cause was found to account for the murmur except a small chorda tendinea running through the ventricle.

The fourth and fifth specimens showed

MULTIPLE SECONDARY GROWTHS.

In the first case the growths were in the myocardium of the right auricle and ventricle, and followed primary carcinoma of the kidney. The lesion had not manifested itself by any physical signs before death.

In the second specimen there were similar growths, mostly in the walls of the left ventricle. The primary cancer was in the left ovary, and was of small size. The patient had died suddenly in an attack of cardiac syncope, there having been no symptoms suggestive of a serious affection previously, with the exception of one or two not very severe attacks of syncope.

The last specimen was one illustrating

ULCERATIVE ENDOCARDITIS,

occurring in a case of pyæmia. Pure cultures of the staphylococcus pyogenes aurens were obtained from the vegetations on the valves, and from necrotic patches in the kidneys.

Dr. G. L. Peabody presented specimens showing a

TRAUMATIC ANEURISM OF THE LEG,

following the lodgement of an embolus, and valvular heart-lesions. The patient was a man, twenty-two years of age, born in England, a nurse by occupation. He gave no

history of any heart-trouble or of any disease ordinarily causing cardiac lesions. He was, according to his own report, perfectly well up to one week before admission to the New York Hospital. At this time he began to be troubled with dyspnœa, and soon after felt a sudden sharp pain in the calf of his right leg. Upon admission an examination showed an increased area of precordial dulness, and there was a distinct mitral systolic murmur and also a double aortic murmur. The pain in the calf steadily increased, and a pulsating tumor appeared in this situation. The patient died from exhaustion without presenting any further symptoms of interest.

Autopsy.—Two hours after death. The right leg and foot were found to be greatly swollen and œdematous.

The pericardium was normal. The heart was large, there was great hypertrophy of the left ventricle, and the muscular tissue was pale and flabby. The valves of the right side were normal. On the ventricular surface of one aortic cusp was a recent vegetation with a base of attachment measuring one-half by one-fourth of an inch. The ventricular surface of the mitral valve was roughened and covered by recent fibrinous masses, which were firmly adherent to the surface of the valve. The surface beneath them was roughened. The free edges of the segments of the mitral valve were ulcerated. Several of the tendinous cords were seen to be completely separated from the cusps. The lungs showed hypostatic pneumonia, besides the interstitial process common in mitral disease.

The spleen was somewhat larger than normal, and contained several old decolorized infarctions.

The only other parts of the body that presented lesions of interest were the tissues of the right leg. In this the femoral and popliteal arteries were normal. A rent was discovered in the posterior tibial artery, near its origin, which established a communication with a large, irregular, ill-defined cavity filled with recently coagulated blood. The inner surface of the walls of this traumatic aneurism was seen to be smooth in some places; in others, it was made up of lacerated muscular tissue. The intermuscular spaces of the calf of the leg were filled with blood, as was also, in some places, the subcutaneous tissue.

The length of the aneurismal sac was twenty centimetres, and its diameter half as great.

Dr. Peabody regarded the case as one of interest from its unusual features, especially the development of the traumatic aneurism following the lodgement of an embolus in the posterior tibial artery, together with the absence of all previous history of heart disease, or of any of the usual causes of heart disease.

Dr. F. T. Hopkins presented a specimen of

INCOMPLETE CLOSURE OF THE FORAMEN OVALE; PATENT DUCTUS ARTERIOSUS.

The patient, Louise F——, came under his care at the New York Foundling Asylum on February 3, 1888, when she was two weeks old. She was suffering from diarrhœa, which had begun two or three days before. She was a small, miserable-looking baby, weighing about five pounds. At this time she was decidedly cyanotic, and the mother stated that she had been a "blue baby" from birth. No signs of pulmonary trouble, and no abnormal cardiac sounds could be detected. There was a slight improvement noticed in the cyanosis upon the cure of the enteritis, but it was only temporary. The cyanosis again deepened, and the child became somewhat stupid, and died on February 9th.

The autopsy was made two days after death by Dr. Northrup. The lungs were fully developed. There was no inflammation, but a moderate hypostasis of both lungs. The heart weighed six drachms. The foramen ovale was in part closed by a semilunar membrane, imperfect in its upper anterior portion, where it was thick and redundant, and retained its fœtal, valvular character. In addition to this, there were two openings in the lower posterior portion of the septum, which had no valvular character, and could not prevent the passage of blood through them. These last two openings were respectively about one and two millimetres in diameter, and allowed the venous blood to pass directly into the left auricle. The ductus arteriosus was pervious, and an ordinary grooved director could easily be passed through it. The heart and great vessels were otherwise normal. The liver was fatty, and weighed four ounces. The spleen weighed half an ounce. The other organs were normal.

The President presented a specimen of

REPTILIAN HEART, OR, RUDIMENTARY AURICULAR SEPTUM, RUDIMENTARY VENTRICULAR SEPTUM, OBLITERATED PULMONARY ORIFICE AND TRUNK, ABSENCE OF PULMONARY VEINS, ABSENCE OF MITRAL ORIFICE, ANOMALOUS VEINS AND ARTERIES.

The child from which this specimen was taken lived thirty-one days; its chief symptom was cyanosis (a "blue-baby"). At death it was found to have one auricle and one ventricle.

A female child, two weeks old, was brought to the New York Foundling Asylum and entered upon the books with the note, "Condition miserable—cyanosis." She remained under observation two weeks, continuing in the same

miserable condition, becoming temporarily less and more cyanotic, and died at the age of one month.

Diagnosis during life was patent foramen ovale and ductus arteriosus.

Autopsy.—The body was small, weighing seven and a half pounds, poorly nourished, and livid, more especially about the head and upper portion of the trunk. The heart inclined to the right side at the angle it usually

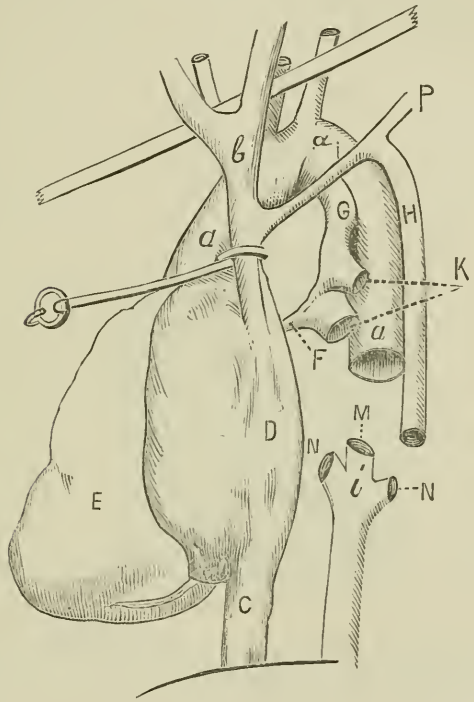

FIG. 1 (life size).—Shows the heart in position. *a*, Aorta; *b*, vena cava superior; *c*, vena cava inferior; *d* and *e*, auricle and ventricle; *f*, rudimentary pulmonary artery; *g*, ductus arteriosus, patent; *h*, vena azygos, anomalous; *i*, gastro-hepatic artery, anomalous; *k*, pulmonary branches from ductus arteriosus; *n*, pulmonary veins; *p*, superior intercostal vein.

bears to the left, the left lung, however, having the usual two lobes. Posterior portions of both lungs were congested; otherwise normal. The pleura, pericardium, and peritoneum contained a small quantity of clear, straw-colored fluid. The heart consisted of one auricle, into which emptied two veins, the superior and inferior venæ cavæ, and one ventricle giving off one artery, the aorta. The ventricular walls were of uniform thickness, showing, without, an intraventricular groove, and within, a ridge of

rudimentary septum. The auricle presented externally the usual outline of two auricles with their appendices. Within there was a thin triangular membrane, having the location and appearance of an enlarged Eustachian valve ; otherwise, there was no attempt at partition. The superior vena cava opened by a valve-like orifice to the left of the rudimentary septum, corresponding nearly to the usual position of the pulmonary veins. The inferior vena cava opened in its normal position at the right of the base of the Eustachian membrane.

The pulmonary artery was represented by a fibrous

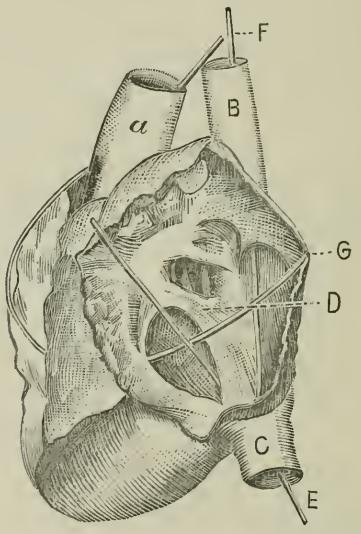

FIG. 2 (life size).—Showing anterior half of auricle laid open. *a*, Aorta ; *b*, vena cava superior, through which passes a probe into left half of auricle ; *c*, vena cava inferior, through which passes probe (*e*) into right half of auricle ; *d*, rudimentary septum or persistent Eustachian valve ; *g*, probe passing through auriculo-ventricular opening and out through aortic orifice.

string attached to the base of the heart in the angle formed by the right auricle and origin of the aorta. Its orifice had no rudimentary trace, and its trunk, as far as the ductus arteriosus, was impervious.

The aorta was given off from the common ventricle in the median line, having upon its right the interventricular groove and rudimentary pulmonary artery. Its relation to the left heart was obviously normal.

The superior vena cava passed down the median line to the left of the aorta, instead of taking its usual course upon its right.

The valves of the heart were two in number, the usual

valves of the aortic orifice and the tricuspid auriculo-ventricular.

Ductus arteriosus Botalli was pervious and equal in calibre to the innominate artery. It descended from beneath the arch of the aorta in a wavy, twisted course for half an inch, when it was joined by the fibrous rudiment of the pulmonary trunk, at this point giving off several branches to each lung.

Below the root of the left lung was a large vessel, receiving blood from the ductus arteriosus by a small straight trunk (Fig. 1, *m*, represented here too large), and from a large vessel coming from each lung. This vessel was without valves, one-third the size of the aorta, conducting arterial blood, and receiving most of its current from the pulmonary veins. It seemed to be physiologically the pulmonary veins, but, instead of emptying into an auricle, it continued downward in an anomalous course, and applied itself to the lesser curvature of the stomach, giving to that organ a rich supply of branches and continuing on to furnish a large trunk to the liver. It was then pulmonary vein and gastro-hepatic artery.

At the usual position of the cœliac axis the aorta gave off a group of arteries as follows: Two renal, superior mesenteric, and splenic.

There were also anomalies among the veins. The left common iliac emptied into the left renal vein. The right common iliac received the blood from the right renal and joined with the left renal at the border of the liver to form with the hepatic vein the inferior vena cava. The azygos veins, major and minor, were represented by an anomalous vessel of large size running along the left side of the aorta, which, having received the superior intercostal, curved forward over the arch of the aorta and emptied into the superior vena cava.

The circulation, then, was maintained by a single heart. The auricle received the customary vessels of the right side; the ventricle discharged its blood into the aorta. At each circuit a portion of the current was turned aside to pass through the lungs, become aërated, pass on to the viscera, return by a short route to the auricle and ventricle, and thence for the most part to pass to the general circulation.

From likeness to the blood-course in lower forms of life, this heart is denominated "reptilian."

The President then presented a second specimen, showing

ANEURISM OF THE INTER-AURICULAR SEPTUM WITH PERVIOUS FORAMEN OVALE.

The patient was a male child, one week old. He was brought to the New York Foundling Asylum, three days

before death, with a history of having been moderately "blue" from birth, and of frequently crying very hard, at which times he became "black in the face." During the remaining days of life he became more and more cyanotic; the temperature of the body fell below normal; the pulse gradually failed; the breathing ceased. At death the contrast of the child's purple skin against the white bed was strongly marked.

At the autopsy the lividity was seen to be most pronounced about the head and upper portion of the trunk and arms. The body was well developed; the umbilical cord undetached. The lungs were fully inflated and normal.

The heart was large, the left ventricle distended, its walls thin. The foramen ovale was pervious, and large enough to allow an ordinary lead-pencil to pass through. This opening was at the anterior upper margin of the fœtal foramen, the remaining portion being filled by a crescentic membrane advancing from attachments upon the entire remaining periphery. This membrane, instead of being tense and tending to contract the orifice, was lax, and, together with the adjacent portion of the auricular septum, became prolonged into an aneurismal sac. The finger-tip, pushed gently against the lax membrane, carried it forward half an inch, the first phalanx being covered as with a glove-finger.

The specimen showed the aneurismal sac, which had been distended; also the aperture, which was as large as the average fœtal foramen, though apparently partially closed by the valve-like margin of the crescentic membrane.

The other organs were normal. The condition of the foramen ovale was believed to be the cause of the cyanosis. The ductus arteriosus was contracted, and allowed little or no blood to pass.

The Society then went into executive session.

Stated Meeting, March 14, 1888.

W. P. Northrup, M.D., President, in the Chair.

Dr. Frank Ferguson presented a specimen of

ANEURISM OF THE ARCH OF THE AORTA,

which had ruptured through the pulmonary artery into the right ventricle. The person from whom the specimen was taken was past middle life, and had died soon after admission to St. Luke's Hospital. The only history obtainable was that of the patient having been troubled with shortness of breath for some time before his admission to the hospital. A peculiarity of the case was that one of the cusps of the pulmonary valve was found to be glued tightly to the wall of the pulmonary artery for a distance of about an inch.

Dr. Ferguson also showed an immense

COLLOID CARCINOMA OF THE PERITONEUM,

removed from the body of a man fifty-one years of age, who had been admitted to the New York Hospital during the service of Dr. Peabody, on July 30, 1887. For the past four years he had had repeated attacks of muscular rheumatism. He had never had malaria, syphilis, nor symptoms of any renal trouble except swelling of the feet, from which he had suffered continuously for a year. Three years before admission his abdomen began to swell, and he had a burning pain about his waist. He also experienced difficulty in urination, the urine being passed at first in a small stream, but finally, the bladder becoming over-distended, incontinence ensued. During this time the patient lost flesh and strength. Examination of the abdomen showed the presence of a large, firm, unyielding mass, tender on pressure, and also of a number of smaller tumors, situated apparently just beneath the abdominal wall. There was no unusual prominence of the superficial abdominal veins. The abdomen measured 105 ctm. in circumference at the level of the lowest ribs.

EXPLORATORY LAPAROTOMY

was performed on August 6th. The parietal peritoneum being divided, the omental tumor was seen presenting, and fluid and colloid material issued from the wound. The patient died two days later.

Autopsy, seven hours after death. The abdomen was enormously distended, tense, and hard on palpation. The entire peritoneum, both visceral and parietal, was infil-

trated with nodular tumors of a pale, translucent material. The coils of small intestine were bound together, partly by adhesions of inflammatory origin, and partly by the union of contiguous masses of tumor in the peritoneum. The omentum was infiltrated and greatly thickened, covering the small intestines like the back of a turtle. The omental tumor measured 25 ctm. from above downward, 45 ctm. from side to side, and was 7 ctm. thick. It weighed 4.1 kilograms. The spleen was enlarged, but appeared to be normal in structure. It was surrounded by a mass continuous with the omentum, measuring 6 ctm. in thickness. The wall of the stomach was infiltrated with tumor masses. Its mucous surface was pale and slightly pigmented in places, but there were no ulcers or abrasions. The orifices of the stomach were normal. The small intestine and mesentery were so matted together that the former could not be opened. The mesentery, in places, was as much as 10 ctm. thick.

Microscopically, the tumor was found to consist of a fibrous stroma, containing large alveoli lined with epithelial cells undergoing colloidal degeneration. It was only in the more recent growths, however, that this structure could be made out, as the sections in general showed simply fibrous material without any definite arrangement.

Dr. Ferguson then presented a heart showing lesions of

ACUTE PERICARDITIS.

The specimen was removed from the body of a man aged eighteen years, who had been admitted to the New York Hospital on January 31, 1888. About two years ago he had had an attack of "rheumatic fever," since which time he had been in apparent good health, except that he was short of breath on exertion, and had had occasionally palpitation of the heart. Six days before admission he had another attack of rheumatism following exposure. Examination showed an enlarged superficial cardiac area to the left, the apex being in the mammary line in the sixth intercostal space. A systolic murmur, not transmitted, was heard at the apex. Over the aortic area was heard a double murmur, the diastolic being the louder of the two. This murmur was transmitted upward into the vessels of the neck. A week later no murmur was audible over the aortic area, but there was a loud bruit in the vessels of the neck. A loud blowing murmur also was now heard at the apex and transmitted toward the axilla. The patient died on February 14th.

Autopsy, seven hours after death. The heart appeared to be very large, it being uncovered by the lungs throughout almost its entire extent. It weighed 430 grams. The pericardium contained 500 c.c. of turbid serum and pus.

The pericardium throughout was covered by recent lymph. The ventricles were dilated and the ventricular walls hypertrophied. The cusps of the aortic valve were thickened and retracted. There was

ATROPHY OF THE TENDINOUS CORDS

of the mitral valve, and one of them had ruptured through ulceration, presenting two club-shaped extremities. There was fat in the muscular tissue of the walls of both ventricles and in the fleshy columns.

The spleen was large, hard, and dark in color. The liver presented the appearances so common in this organ in cardiac disease. The kidneys were large and congested, and the cortices swollen and opaque.

Dr. Van Santvoord said that he had a heart which he had been intending to present to the Society for some time, in which there were lesions of the chordæ tendineæ very similar to these. During life a loud and musical murmur had been heard. The patient had died in Randall's Island hospital, but unfortunately no previous history had been obtainable. He attributed the musical character of the murmur to the evenness of the lesions.

The President referred to the heart presented at the last meeting by Dr. Biggs, in which a systolic musical murmur, heard at the apex, was produced by a chorda tendinea running through the ventricle.

Dr. Ira Van Gieson presented some microscopical specimens illustrating

FAT-NECROSIS IN THE PANCREAS.

The specimens were taken from a woman aged eighty. In the superior border of the pancreas, about midway between the head and tail, was a soft, yellowish-white nodule, about five mm. in diameter, having a degenerated pasty centre. In the tail of the organ was a similar superficial nodule the size of a pin's head. A portion of the soft centre of the larger nodule examined microscopically in the fresh condition, consisted of larger and smaller, rather opaque globules, some of which stained with osmic acid. In sections through the nodules, amorphous masses were seen in places partially filling up the fat-spaces; in other places the degenerated fat-cells had coalesced, forming irregularly shaped necrotic masses, which stained brightly with eosin. The lesion seemed to be produced by the coalescence of necrotic fat-cells. Some of the acini of the pancreas contained hyaline cast-like bodies surrounded by the compressed cells of the acinus. Fat-necrosis has been studied by Ponfick (*Virchow's Archiv*, 56, p. 541), Chiari (*Prag. Wochenschrift*, 1883, 30–31), and Balser (*Virchow's Archiv*, 90, p. 520). The latter

author found the lesion present in the pancreas in five instances in twenty-five autopsies, and recorded a case in which the lesion occurred in the omental fat, and extensively in the pancreas, which was the seat of fatal hemorrhage. Balser is disposed to think in this case that the fat-necrosis, by producing the pancreatic hemorrhage, was the indirect cause of death.

Dr. Ferguson said that he had made it a practice to examine the pancreas at his autopsies for some time past, and he had come to the conclusion that fatty necrosis of this organ was nearly always present. The fat might be found in the cells, evenly distributed throughout the pancreas. This occurred usually, though not always, associated with parenchymatous degeneration of other organs. Then again, the pancreatic cells might be unaffected, while large masses of what seemed to be fat was found in the interior of the lobules, the pancreatic tissue being destroyed. These masses were of a brownish color, and were frequently of sufficient size to be seen by the unaided eye. More striking, however, on gross inspection was the accumulation of fat along the planes of fibrous tissue, which might be so great as to cause destruction of the lobules by pressure.

When the masses within the lobules were treated with a weak caustic alkali, they sometimes disappeared. At times the brownish amorphous material on the outside disappeared on the application of weak acids, and bodies having the appearance of fat remained. These were, however, only in part soluble in ether, and what remained seemed to be leucin. He did not think that fat necrosis of the pancreas was the cause of hemorrhage in the organ, as he had never seen any evidence of this in his autopsies. He did not know why the fat was generally brownish in color, unless it was a result of the action of the secretion of the gland.

Dr. Van Gieson said that he was not inclined to believe that there was any causal relation between fat-necrosis and hemorrhage. These areas of fat-necrosis were observed as small, soft, circumscribed masses, but were not at all the same as lipomatous growths. Fat-necrosis was not always confined to the pancreas, and the speaker believed that it had also been observed in the form of little nodules in the peritoneum.

The President asked whether there were any gross appearances in the pancreas which would lead one to suspect the existence of fat-necrosis.

Dr. Van Gieson replied that, when extensive, it could be recognized by the presence of the brownish nodules, but in many cases these nodules were microscopical in size.

The Society then went into executive session.

Stated Meeting, March 28, 1888.

W. P. Northrup, M.D., President, in the Chair.

Dr. Eugene Hodenpyle presented a specimen of

EPITHELIOMA OF THE ŒSOPHAGUS,

with perforation into the posterior mediastinum, gangrene of the right lung, and acute pericarditis.

F. S——, aged thirty-six. Family and previous history negative. The patient stated that up to four weeks before his admission he was in good health. At that time he began to have difficulty in swallowing, and attempts to swallow solid food were frequently followed by regurgitation. When first seen he was able to swallow liquid food only, but a full-size bougie was passed into the stomach without detecting an obstruction. He rapidly became very much emaciated. The dysphagia became more and more marked, so that toward the close of the disease it was almost complete. Two days before his death it was noticed that the patient's breath was extremely offensive, and the presence of fluid in the right pleural cavity was made out. He died, January 27th, about two months after the first symptoms of his disease were noticed.

Autopsy.—The left lung was emphysematous, and the right lung had but two lobes. The pulmonary pleura was covered by a layer of dirty yellow fibrin, and in the pleural cavity there was about one quart of greenish-yellow serum, while at the base of the lung was a sacculated accumulation of clear serum. The lower lobe was consolidated, of a grayish-brown color, and granular on the cut surface. The upper lobe was collapsed. At the hilus of the lung was seen a large irregular cavity, with ragged edges, filled with a very offensive black fluid.

The pericardial sack contained two ounces of bloody serum, and both surfaces of the pericardium were coated with a thin layer of fibrin. The heart was otherwise normal.

At the cardiac end of the œsophagus was a large, raised, ulcerated mass, four inches in length, two inches and a half in width, and three-fourths of an inch in thickness.

It contained a slit-like perforation at its centre communicating with the posterior mediastinum. The calibre of the œsophagus was but little, if at all, constricted by the new-growth. In the posterior mediastinum the bronchial glands were enlarged and softened, and the surrounding structures were partially gangrenous.

Microscopical examination of the new-growth showed it to be an epithelioma containing a number of giant cells. The pneumonic area of the lung showed necrotic changes in places.

Dr. Hodenpyle also presented some specimens removed from the body of a man who was supposed to have been suffering from

PSEUDO-LEUKÆMIA.

The patient was sixty-two years of age. He was admitted into St. Francis Hospital on January 31, 1888, with a history of repeated attacks, during several years past, of diarrhœa, each attack lasting usually from one to three weeks. He had also had chronic bronchitis. He denied excessive drinking, and had never had rheumatism or malarial fever.

On admission to hospital he was examined for evidences of syphilis, with negative results. The inguinal, cervical, and epitrochlear glands were considerably enlarged and somewhat tender. The patient said that five weeks before his admission he had an attack of diarrhœa which had lasted up to the present time. He was now having about twenty loose passages a day, accompanied by some rectal tenesmus. Three weeks later his feet and legs became swollen. When first seen he was greatly emaciated and somewhat jaundiced. He complained of pain all over the abdomen, which was considerably distended with gas. No fluid was detected in it. The area of splenic dulness was increased. The liver was normal in size. The lungs showed lesions of general emphysema and bronchitis. The heart was normal. The urine contained albumen and casts. Pulse, 90 and small; respiration, 24; temperature, 99° F. Appetite good. No vomiting.

The patient became more and more emaciated, and finally died of pneumonia on February 16th.

Autopsy, eighteen hours after death. Lungs: There were slight adhesions of both lungs to the costal pleura, and one quart of clear serum was found in the left pleural cavity. On the pleura of both lungs were numerous white pin-head patches, not elevated and mostly in the centre of pigmented portions (multiple fibromata?). Lungs emphysematous, œdematous, and congested posteriorly; bronchi thickened and filled with muco-purulent fluid. There was little fluid in the pericardial sac. The heart-muscle was pale and flabby; the aorta was atheromatous.

There was no fluid in the abdominal cavity. The liver was somewhat enlarged, pale, and friable, and on cut section showed irregular yellowish-white, coarser, and finer mottling, and narrow irregular, whitish streaks.

The spleen was 16 ctm. long, 8 ctm. wide, and 7 ctm.

thick; its surface roughened by patches of perisplenitis. The organ was hard, dark red in color, mottled with small irregular, whitish streaks and spots, and showed several dense, white, hard, irregular, wedge-shaped masses presenting the appearance of white infarctions. Some of these infarctions were red in the periphery and some were of reddish color throughout.

The pancreas was enlarged and hard.

The kidneys were of normal size and consistence. The left contained a small cyst. Cortex slightly thickened. Markings indistinct. Capsules adherent.

The supra-renal capsules were hard.

The solitary glands of the stomach, especially those about the pylorus, were swollen, and near this orifice was a soft tumor about two centimetres in diameter. The small intestine contained swollen solitary follicles and Peyer's patches.

In the large intestine, the solitary follicles were seen to be considerably swollen, the centres being depressed and blackened. Scattered about in the mucous membrane were a large number of spheroidal or more or less irregular nodules, occasionally pedunculated, varying from one-half c.c. to two or three c.c. in size.

The mesenteric glands were enlarged and soft.

Behind the peritoneum and surrounding, but not involving, the great vessels, was a large, hard, yellowish-white mass, extending from almost up to the diaphragm above into the pelvis below, where it had partially surrounded the sigmoid flexure of the colon, which was displaced toward the middle line.

The bladder was normal.

Microscopical examination.—The masses in the large intestine and those behind the peritoneum and in the stomach had essentially the same structure, being composed of large numbers of small spheroidal cells closely packed in a scanty connective-tissue reticulum, blood-vessels, etc. The enlarged mesenteric glands showed the lesions of hyperplasia.

The kidneys showed chronic diffuse nephritis, and the blood-vessels were partially filled with spheroidal cells.

The liver was thickly studded with masses similar in appearance to the tissue in the tumors of the intestine. Around the masses the liver-cells were flattened, pigmented, and fatty. The blood-vessels contained numbers of spheroidal cells. A similar condition was seen in the supra-renal capsules and the tissues surrounding them.

The spleen showed the usual appearances of white and red infarctions. There were enlarged Malpighian bodies and a great overcrowding of the cavernous veins with small spheroidal cells.

Dr. Prudden remarked upon the number and unusual size of the tumors in the large intestine. It was not known in this instance whether there was any increase in the number of the white blood-cells or not, and it could not be determined, therefore, with certainty whether the case was one of leukæmia or of pseudo-leukæmia.

Dr. Mendelson asked whether the spheroidal cells referred to were white blood-corpuscles or not.

Dr. Hodenpyle thought that they were.

Dr. H. M. Biggs presented a number of specimens illustrating

DISEASES OF THE CÆCUM AND VERMIFORM APPENDIX.

The first specimen was from a case of general peritonitis, following

SLOUGHING OF THE TIP OF THE VERMIFORM APPENDIX,

with the following history: The patient, a young man about thirty-three years of age, was seized at night with vomiting and diarrhœa, attended by marked abdominal pain. He had been well before this time. The physician who was called to see him supposed he had to deal with an ordinary case of gastro-enteritis, and treated him accordingly. Although he temporarily improved, he soon grew worse, the vomiting continued, and finally became fecal in character. Dr. Bryant was asked to see the case in consultation with Dr. Charles Simmons, the physician then in charge. At that time the abdomen was tensely distended and tympanitic. Patient had only a moderate elevation of temperature, a rather rapid pulse, fecal vomiting, and severe pain referred to the epigastrium. The tenderness was not very marked and was not localized. A diagnosis of general peritonitis, due probably to perforation of the vermiform, was made. The patient gradually sank and died.

At the autopsy a general peritonitis was found, with great matting together of the coils of intestines and considerable thick fibrino-purulent exudation. Scattered here and there between the coils of intestines were found numerous small pockets of pus. After a very careful search, the stump of the vermiform was found with its end sloughed off. In the peritoneal cavity was a small hard, fecal mass, which had evidently been the cause of the trouble. There was no apparent attempt at the incapsulation of the exudation around the cæcum or localization of the process. The vermiform projected from the inner side of the cæcum freely into the peritoneal cavity.

The second case, seen with Dr. L. M. Yale, was also one of vermiform sloughing, with

FECAL MASSES IN THE PERITONEUM.

The main points in the history were as follows: A boy, sixteen years of age, came home from school feeling unwell, and remained at home for this reason. He was first seen February 4th, but was taken sick two or three days before, at night, with severe abdominal pain. When first seen he had some elevation of temperature, abdominal tenderness, and slight tympanites. There were exaggerated nervous symptoms present at this time that rendered an immediate absolute diagnosis impossible. The patient was again seen at 6 P.M. on the same day, and a diagnosis of general peritonitis, probably following vermiform perforation, was made. Dr. Janeway saw him the same evening, and agreed with the diagnosis. There was no localized pain or tenderness. Dr. Sands saw the boy the following day, and made the same diagnosis. Patient died five days later. There was occasional vomiting during the sickness. Toward the end all abdominal tenderness disappeared.

At the autopsy, a general peritonitis was found. The coils of intestines were firmly matted together, and a moderate amount of fibrino-purulent exudation was found in the peritoneal cavity. The last four inches of the vermiform had sloughed off and lay loosely in the peritoneal cavity. This contained three small hard, fecal masses.

Lying near it in the abdominal cavity was another large hard, fecal mass, that had apparently been situated at the point of sloughing. The vermiform had evidently extended freely downward and inward. There was no apparent attempt at a localization of the process.

Dr. Biggs then presented a third specimen, showing

PERFORATION OF THE VERMIFORM APPENDIX.

The case was seen with Dr. W. T. Jenkins, Deputy Coroner. The death was reported by the physician in charge as having resulted from general peritonitis following the production of abortion. There was no history of an abortion, but the symptoms and condition aroused the suspicions of the physician in charge. The patient was a servant-girl, aged twenty-five. She was suddenly seized with severe abdominal pain during her menstrual period. The physician who was called to see her made a diagnosis of general peritonitis, and, from the presence of metrorrhagia, the fact that the girl was unmarried, and some evidence of a circumstantial character, concluded that the case was one of criminal abortion, and he so reported it to the Health Board. The autopsy revealed the presence of

general peritonitis resulting from perforation of the vermiform appendix. A small mass of hard fecal matter was found in the peritoneal cavity. The vermiform extended freely out into the peritoneal cavity, and the point of ulceration, which was about one and a half inch from its origin, was only partially covered by peritoneal exudation.

Another specimen shown was one of a

LONG VERMIFORM APPENDIX,

measuring nearly nine inches in the fresh condition. It arose just below the ilio-cæcal valve, passed directly upward behind the iliac mesentery, being extra-peritoneal until just above the beginning of the mesentery, when it became intra-peritoneal, and continued its course up inside of the ascending colon as far as the under-surface of the liver, when it turned forward, lying underneath the liver.

In the next specimen there was

OBLITERATION OF THE OPENING OF THE VERMIFORM

just at its origin. Following this, an accumulation of the secretion from the mucous membrane lining occurred and produced an enormous dilatation. In the fresh condition it was considerably more than an inch in diameter and about four inches in length. There were no symptoms (so far as known) during life resulting from this condition.

The next specimen was one of

PERITYPHLITIS WITH PERFORATION INTO THE ASCENDING COLON.

The clinical history of the case was rather obscure. It was supposed to be a case of psoas abscess for some time. Pus was passed by the bowel. At the autopsy there was found a large abscess-cavity extending far up along the ascending colon and displacing it to the left, with extensive induration in the connective tissue. After a careful search, a communication between the cavity and the ascending colon was discovered. This was situated just below the pelvis of the right kidney.

Dr. Biggs then reported another case,

RUPTURE OF THE RECTUM,

the specimen of which he was unable to present at the time. The patient had suffered for a long time with symptoms of intestinal obstruction, supposed to be due to stricture situated at the upper portion of the rectum, or in the sigmoid flexure. Patient had not had a movement for about ten days when, after various means had been tried to move the bowels, a manual examination of the rectum was made under ether, and an enema of ox-gall was afterward given. The patient complained of severe

abdominal pain after coming out from ether, soon went into collapse, and died twenty-four hours later.

At the autopsy a rupture through the anterior wall of the rectum was found, about four inches in length, and the ox-gall enema was mingled with the inflammatory exudation following a general peritonitis. At the junction of the descending colon with the sigmoid flexure was found a slight stricture with a large ulcer, the base of which was formed by the peritoneum. Surrounding it in the peritoneal cavity, loosely incapsulated by the agglutination of the coils of the intestines by the exudation, was a small pocket of pus. On the inner surface of the cæcum, just above the base of the vermiform appendix, was a second ulcer that had extended through the peritoneum, and which opened into a small intra-peritoneal pocket of pus, similarly incapsulated by the agglutination of the surfaces of the peritoneum. These two pockets of pus would probably both have opened soon into the abdominal cavity and produced a general peritonitis, had not the rupture of the rectum and the ox-gall enema earlier completed the clinical history of the case. The ulcers were apparently simple, and the remainder of the mucous membrane of the alimentary tract was entirely free from disease.

The last specimen was one of

IMPACTION OF A FOREIGN BODY IN THE VERMIFORM APPENDIX.

It was found accidentally at an autopsy when, so far as had been known, there had been no symptoms of any kind referable to this region. It was firmly fixed in the appendix, and the history of the other cases would have probably been repeated in this, had not an intercurrent disease cut short the life of the patient.

Dr. Biggs then concluded as follows: "During the last two years I have examined and noted, in the autopsy- and dissecting-room, the relation, condition, position, etc., of the vermiform appendix in nearly two hundred cases. I have also had a number of autopsies on other cases quite similar in all respects to those described, and, as a result of these examinations, I have come to the conclusion that, in a very large proportion of the cases of general peritonitis following ulceration and perforation of the vermiform appendix, the condition was the result of the impaction of a large foreign body in an appendix that extended freely into the peritoneal cavity, having a long mesentery and being easily movable. It is under such conditions that there are present the most favorable influences for the expulsion of any foreign matter that may make its way into the appendix. When thus situated the vermiform is moved about freely in the abdomi-

nal cavity by the peristaltic movement of the intestines, and through this constant change in position its own muscular walls are most favorably placed for expelling any matter that may make its way into its lumen. Consequently, impaction only occurs when a body of considerable size (so large that it cannot be expelled) is formed, or finds its way into the cavity. Thus the same conditions that at first made such a vermiform less liable to disease now place it under the most unfavorable conditions, so far as the patient is concerned. For the constant motion of the intestines prevents the formation of any adhesion around the appendix as the ulceration extends through its walls, and, finally, when perforation occurs, a direct communication with the abdominal cavity is at once established, and a general peritonitis is the result. I believe further that in most cases, at least where general peritonitis follows vermiform perforation, there is a direct communication with the peritoneal cavity from the first. It does not follow secondarily from rupture of an abscess into the cavity, and any operative interference, to be successful, must be early resorted to.

"As further evidence that this is the case, I would note that in cases where the vermiform is freely movable there are rarely evidences of disease or changes in its mucous membrane; while, on the other hand, when the appendix has a short mesentery, or no mesentery at all, or lies back of the cæcum coiled up and fixed in its position, or when the end is bound down at an angle with the body, there are very frequently evidences of disease in the mucous membrane. It is in such cases that there is the greatest difficulty in expelling any foreign matter that may make its way into it, and it is also in such cases that disease with ulceration, etc., is most frequent. But under these conditions the appendix is practically immovable, and before perforation occurs adhesions have formed that shut off the abdominal cavity, the process extends out into the peri-cæcal connective tissue, and perityphlitis results. Consequently this is a far more frequent result of disease of the appendix than general peritonitis. I would not, however, be understood as inferring that none of these cases result from disease of the cæcum, for some of both forms undoubtedly have their starting-point here.

"As an interesting point in the clinical history of the reported cases of general peritonitis following perforation of the appendix, I would call attention to the absence of local pain and tenderness, and the reflection of the pain to the epigastric region."

Dr. Biggs also showed a specimen of rupture of the left broad ligament, occurring in a case of criminal abor-

tion. The greater part of the fœtus, which was at about the fourth month, lay between the layers of the broad ligament. The rupture of the uterus had begun about three-fourths of an inch above the external os.

Dr. Hamilton presented a specimen of

MULTIPLE DIVERTICULA OF THE SIGMOID FLEXURE,

removed from a man aged forty-five, who had died of chronic phthisis. The small intestine was normal. Lying on the outer surface of the sigmoid flexure, between the longitudinal muscular bands, were about a dozen spherical bodies, varying in diameter from one-sixteenth to one-half inch. On cutting into one of them it was seen to have a thin wall and to contain hard fecal matter; it communicated by a narrow channel with the interior of the intestine.

A microscopic examination of one of these diverticula showed that its wall was similar to that of the sigmoid flexure, but that the mucous membrane was compressed and flattened by the fecal contents, and the muscular layer was quite thin, and in places entirely wanting. It was evident that they were formed by a protrusion of the mucosa through the muscular coat. Diverticula in various portions of the intestines are recorded in pathological literature. Dr. Hamilton thought that the case was noteworthy for several reasons: First, the lesion was in the sigmoid flexure, where large fecal masses may accumulate. Second, a complete analogy existed between this lesion and diverticula of the bladder. Lastly, a possible result of such a lesion might be perforation, producing peritonitis and death.

Dr. Mary A. D. Jones presented two specimens of

TUMORS REMOVED FROM THE PELVIS.

The first patient gave a history of abdominal pain and distress extending over many years, and unrelieved by treatment. In fact, so much did the patient suffer that she said, the day before the operation, that if she was told that she had but one chance in twenty-five of recovery she would take that chance and have the operation. Examination showed the uterus pulled to the extreme right by shortening of the right broad ligament, and in the centre of the floor of the pelvis a soft tumor, exquisitely painful and evidently the seat of fluctuation. A diagnosis of an ovary enlarged by an abscess was made. The patient was successfully operated on by abdominal section, and both ovaries and tubes removed.

The specimen exhibited showed a portion of the left ovary to be of a hard, nodular, cheesy consistency, and the rest of it converted into a large pus-cavity, a portion

of whose walls was almost of the thinness of paper; the danger of rupture, with possibly serious results, being obvious. Both tubes were in a state of chronic salpingitis. The right ovary was one-fourth of normal size and cirrhotic.

The second patient also had a fluctuating tumor in the pelvis adherent by strong adhesions. A first examination gave the impression of an enlarged and extremely retroflexed uterus, and so it had been diagnosed; but its softness and the sound passing into the uterus in the normal direction made the presence of an enlarged tube very probable. On operation the tube was found to be of the size of an orange, and completely adherent to the posterior part of the uterus and the wall of the pelvis. After separation of the adhesions, and as the mass was being lifted out of the abdominal cavity, it ruptured and quantities of dark clotted blood escaped. The patient made a good recovery.

The specimens showed the left tube to be closed at its distal end, and enlarged to three and a half inches in diameter and filled with blood; it was adherent to the left ovary. The right appendages were also bound down by adhesions, the tube adherent to the ovary and closed at its fimbriated extremity. Diagnosis, pyo-salpingitis.

Stated Meeting, April 11, 1888.

W. P. Northrup, M.D., President, in the Chair.

The President presented, on behalf of candidates, a specimen of *Acute Suppurative Arthritis following Scarlatina*, and one of *Supernumerary Finger*.

Dr. Hodenpyle presented specimens showing

CARCINOMA OF THE PERITONEUM

and carcinomatous nodules in the liver, spleen, and body of the uterus.

Mrs. O——, aged forty, widow two years. Family history negative. She was married at the age of seventeen, up to which time she had always been healthy. She had had two children, the last thirteen years ago. No miscarriages. Menstruation was regular up to the age of thirty-four years, when it ceased. Since then she suffered for a considerable time, in place of her regular monthly periods, with difficult, painful, and frequent micturition, severe pain in the back, and slight swelling of the abdomen. In July, 1887, she was taken with pretty severe pains in the hypogastrium, coming on in attacks lasting about twenty minutes. In September of the same year she noticed a "lump" in the right iliac fossa which gradually grew in size, and was accompanied by swelling of the abdomen. She was then admitted into the New York Hospital, where she says she was examined under ether, that when she came out from the ether her abdomen was tightly bandaged, and when this was removed there was seen a small, nodular, soft, painful growth protruding from the umbilicus. She was a good deal emaciated, and a microscopical examination of the growth at the umbilicus showed it to be an epithelioma. Abdomen moderately distended. Liver normal in size. No splenic dulness. There was a semi-fluctuating mass in the right iliac fossa, three inches in diameter. There was also a large flat mass in the centre of the abdomen, to which the growth at the umbilicus seemed to be connected. The inguinal glands were enlarged. Upon examination per vaginam, the body of the uterus and the ovaries were found to be converted into a large, hard, dense mass. The patient died exhausted April 8, 1888.

Autopsy.—Rigor mortis not well developed. Emaciation extreme. Moderate anasarca of left leg. The right pleural cavity contained a small amount of clear fluid. The right lung was large and œdematous, and contained three grayish nodules about two cubic centimetres in size.

The left lung was small, being compressed toward the vertebral column by an accumulation of clear serum, inclosed by a thick, rather soft, capsule. The fluid within was contained in a number of thin-walled sacs. Bronchial glands enlarged and very soft. The adjacent pericardium was greatly thickened. Heart was very small, otherwise did not appear to be abnormal. The abdomen contained a small amount of clear fluid. The growth at the umbilicus was continuous with a large irregular mass beneath the peritoneum, to which were attached the greater omentum and portions of the intestine. The diaphragm covering the liver was greatly thickened, and the liver immediately beneath was converted into a zone of yellowish-white tissue, about one centimetre in thickness, and considerably softer than the surrounding liver tissue. Scattered through the viscus were several large whitish masses, and the rest of the organ was moderately fatty. The spleen was soft, and contained a large tumor-mass. Pancreas, suprarenals, and kidneys showed no lesions. On the external surface of the gall-bladder, mesentery, intestines, and parietal layer of the peritoneum were a very great number of larger and smaller spherical or irregular masses. In the pelvic cavity was a large mass, partly cystic, on both sides of the uterus, the body of which was greatly enlarged, the cervix and endometrium being fairly normal. The bladder was normal. The ovaries could not be made out from the tumor-mass. The inguinal glands were greatly enlarged and thickened.

Dr. Lewis Hall Sayre presented a specimen of

ROUND CELL SARCOMA

of the arm, removed from a child the day before. The child had always been in perfect health until six years ago, when the mother noticed a small painless swelling on the anterior aspect of the forearm. Various diagnoses were made, and the treatment varied accordingly. Finally, an elastic armlet was applied and worn for four years, but was then discarded because of the discomfort which it caused. For the past two years there had been no treatment. The arm gradually increased in size, more rapidly during the past eight weeks, and measured, when Dr. Sayre saw the case, nine and one-half inches in its greatest circumference. It was distended cylindrically, and was moderately hard to the touch. There were no nodules to be detected, and there had never been any pain from the commencement.

An incision was first made, but it was seen to be impossible to remove the tumor, and the arm was therefore amputated. The new growth, which was attached to the periosteum of both radius and ulna, seemed to be a round-

cell sarcoma. Dr. Sayre requested that the specimen be referred to the Microscopical Committee.

Dr. James E. Newcomb presented the spleen and liver removed from a patient dead of

SPLENIC LEUCOCYTHÆMIA,

with the following history: "Mrs. M——, aged forty-three, born in New York City, of Scotch parentage. One brother died of visceral cancer; family history otherwise negative. The patient was always in good health till the commencement of her fatal illness. Had been married twenty years, and had had three children, all of whom are living and healthy.

"In May, 1885, she had an acute malarial attack, and was in bed six or seven weeks. The disease was intermittent; at first quotidian, but later tertian. There was marked enlargement of the spleen. On getting up in July, the patient noticed this splenic tumor in the left side. At times it was painful, and she had great distress in the epigastric and right hypochondriac regions. There was no stomach disturbance and no other glands were enlarged. She came under my care in July, 1886. She was markedly emaciated, though free from any definite cachectic appearance. She had had considerable epistaxis in the previous few weeks. Menstruation had ceased at the time of the first malarial onset in 1885, and was thereafter present only irregularly. At no time was there hemorrhage from any mucous membrane other than the nasal. No urinary symptoms.

"At this time the abdomen was distended with a mass, hard at some points and fluctuating at others, and resembling on palpation an ovarian fibro-cyst. No distinct areas of splenic or hepatic dulness could be obtained. She continued to emaciate. The fluid in the abdomen was mostly absorbed. About November, 1887, an examination of the blood was made by Dr. W. B. James, who reported that it presented the appearances characteristic of leucocythæmia. At this time it was possible to map out the spleen with great distinctness; it commenced posteriorly at the usual line, and projected forward somewhat across the median abdominal line, and was considerably more than a hand's-breadth in width. The liver flatness ran down to the level of the umbilicus. There was no evidence of fluid in the abdomen.

"These, then, were the four features of the case: The splenic and hepatic enlargements, the blood changes, the emaciation, and the usual symptoms of anæmia. On February 11, 1888, the patient had a fresh epistaxis, continuing at intervals till her death. She complained greatly of præcordial pain. Was seen on the 13th, and an attempt

made to plug the nares with styptic cotton. Only temporary relief was afforded. At noon, on February 14th, she suddenly died.

"As soon as a definite diagnosis was made, she was placed upon Fowler's solution in addition to her previous tonics. None of the remedies employed seemed to be of the slightest service in retarding the progress of the disease.

"*Autopsy*, five hours after death, Dr. James assisting.—Permission was given only to open the abdomen and remove the 'tumors.' The liver measurements were as follows: Transverse, 31 ctm. (12½ inches); antero-posterior, 16 ctm. (6½ inches). In thickness (back part, right lobe), 7½ ctm. (3 inches). Weight, 2,624 gms. (5 lbs. 2 oz.). The measurements of the spleen were: Length, 26 ctm. (10½ inches); breadth, 13½ ctm. (5½ inches); thickness, 6 ctm. (2½ inches); weight, 1,888 gms. (3 lbs. 11 oz.). The liver seemed rather soft in consistency, and the spleen was very hard. There was no enlargement of the retro-peritoneal or mesenteric glands. No evidences of peritonitis; no fluid; kidneys apparently normal."

Dr. Van Gieson showed some specimens illustrating

ACUTE LOCALIZED MENINGITIS, PYLORIC CARCINOMA, AND HYDRO-NEPHROSIS.

The specimens were taken from a strong man, aged thirty-five. Sixteen months before death the man began to have symptoms of gastric and hepatic disturbances, apparently due to the protracted ingestion of alcohol. There was morning vomiting, general emaciation, jaundice, and inability to take food well at times; but severe gastric pain was absent, and carcinoma of the stomach was not decided upon until after a physical examination, three months before death. Five days before death symptoms of meningitis occurred, with violent mania and continuous muscular tremor confined to the legs.

At the autopsy the surface of the pia was dry; on either side of the longitudinal incision there were two nearly symmetrically situated, raised yellow pellicles (3½ by 2 ctm. in diameter), involving the para-central lobules. Nearly surrounding the pylorus, involving diffusely all of the coats of the stomach, was a dense, whitish mass, about 4 ctm. in diameter and 1 ctm. thick. The left hepatic lobe contained several small, hard, white nodules. The left kidney was large and white. The pelvis of the right kidney was dilated, containing 240 c.c. of acid, turbid albuminous fluid, in which were many pus-cells and a few large granular, polymorphous, nucleated cells.

Dr. H. J. Boldt presented specimens showing a

SUPPURATING OVARIAN CYST IN A CHILD,

complicated by acute meningitis. The patient was aged four years. Eight months ago the parents noticed a tumor in the lower part of the abdomen, which gradually increased in size. There was no pain up to within three weeks, when the tumor became very tender on pressure, and the child seemed to suffer pain in the abdomen. One week ago the child suddenly grew worse, became feverish and restless. When first seen by Dr. Boldt it had a temperature of 103.4° F., the restlessness had given way to apathy, and the general picture was that of meningitis. The introduction of an exploring needle into the tumor, which extended above the umbilicus, showed the contents to be pus. It now became a question whether the cerebral symptoms were due to meningitis, or whether they were to be attributed to the presence of the suppurating tumor. In view of the possibility that the latter was the case, an operation was done without delay. The tumor was found to be a simple ovarian cyst, springing from the left side, and containing from one and a half to two quarts of pus. The sac was adherent to such an extent that removal was out of the question, so the walls were stitched to the abdominal wound, the cavity was washed out, and drainage was provided for. The patient did not improve after the operation, making it clear that the meningitis was the main complication, and this was borne out by the condition found at the autopsy. Death occurred two days after the operation. The interesting features of the case were the age of the patient with a suppurating cyst of the ovary, and the complicating meningitis.

Dr. Myers presented some specimens removed from the body of a child nine years of age, who had died from

STRANGULATED INGUINAL HERNIA.

The points in the history of the case, for which he was indebted to Dr. F. T. Hopkins, were as follows: The child had a congenital hernia. One evening the nurse noticed a dark-colored swelling in the groin, but did not think it had any significance and said nothing about it. The child soon after began to show evidences of intestinal obstruction; there were no movements from the bowels, the temperature was high, and there were a rapid pulse and shallow respiration. The child was seen at this time, and the question now was whether there was gangrene or not. It was thought that there was probably no gangrene, as the symptoms of strangulation had existed for so short a time, and it was therefore decided not to operate.

Taxis was tried and the tumor was readily reduced. The child at first improved, but in about four hours symptoms of collapse appeared, and death supervened.

At the autopsy there was seen to be gangrene of the parts involved in the strangulation. The hernia was on the right side, and included a small part of the ileum, the cæcum, and the vermiform appendix. The case was particularly interesting from the fact that gangrene of the gut had occurred in a child in so short a time after the strangulation.

Dr. Frank H. Hopkins presented some sections of a

SUPERNUMERARY FINGER.

The specimen, from which the section was taken, was removed from a mulatto child, a female, at the age of three weeks. The fifth finger grew from the soft parts on the outer aspect of the first phalanx of the little finger of the right hand. It was attached by a slender pedicle, which allowed free movement. The pedicle was tied and the body snipped off. The finger thus removed was one centimetre long, half a centimetre thick, and had a well-developed nail and a joint. The microscope slide herewith presented showed a longitudinal section through the pedicle, through the cartilages, new bone, and joint. The points of interest were : 1. The intra-cartilaginous ossification in the distal extremity of the terminal phalanx. The specimen shows the new bone, new vessels tunnelling into the cartilage, osteoblasts, columnar arrangement of cartilage-cells in the region of the advancing vessels. 2. The joint in early life. 3. Vertical section of nail in its entire length. 4. Pacinian bodies.

The Society then went into executive session.

Stated Meeting, April 25, 1888.

W. P. Northrup, M.D., President, in the Chair.

Dr. H. M. Biggs presented, on behalf of a candidate, specimens of *Carcinoma of the Stomach* with secondary growths in other organs.

The President presented, on behalf of candidates, a specimen of *Lupus Vulgaris Nasi*, and one of *Fluctuating Mammæ in a boy* aged seven days. The glands contained milk.

Dr. Biggs reported from the Microscopical Committee that the specimen shown at the last meeting by Dr. Lewis Hall Sayre was one of round-cell sarcoma growing from the periosteum.

Dr. C. R. Hexamer presented a specimen of

ANEURISM INVOLVING THE THORACIC AND ABDOMINAL AORTA,

removed from the body of a man seventy-three years of age, who had entered St. Francis' Hospital a few days after the blizzard of March 12th, complaining only of exhaustion from exposure, and seeking rest for a few days to recuperate. He was remarkably robust-looking for his age, and was in good physical condition. He said he had not been sick a day for forty years. The heart-action was very feeble, and a bruit was heard both anteriorly and posteriorly. One day, without any premonitory symptoms, the man suddenly had an attack of syncope, went into collapse, and died in about twenty minutes. At the autopsy an aneurism was found of the arch of the aorta, and another of very large size on the abdominal aorta. The latter filled one-half of the abdominal cavity, the sac being eight or nine inches in length by four inches in diameter. The interesting points were the large size of the sac, the double lesion, and absence of all symptoms pointing to the presence of aneurism.

Dr. Mary A. Dixon Jones presented a microscopical specimen of

SARCOMA OF THE CERVIX UTERI.

The specimen was presented not so much to demonstrate the sarcoma as to show that the cervical portion of the uterus was hyperplastic before the invasion of the sarcoma, and that the protoplasm of the sarcoma first invaded the newly formed fibrous connective tissue. This verified the assertion of Thomas Addis Emmet, that cervical lacerations may be the cause of, or invite, malignant growths.

Dr. Jones presented a second specimen, showing an

OVARIAN CYST AND CIRRHOTIC OVARY,

removed by operation from the body of a woman, twenty-two years of age, who had been sick for the last five years, during the whole of which time she said she had not had a well day or one free from pain. She had been under the care of several physicians, and apparently the trouble was an extreme retroversion of the uterus, the organ being so extremely sensitive that it resisted all efforts at replacement. By manual examination a large sensitive mass could be detected in front of the uterus and adherent. She was under treatment for some time in the Woman's Hospital of Brooklyn, but without obtaining any relief. Four days ago an abdominal section was made, and a growing ovarian cyst, three inches in diameter, was found in front of the uterus, and bound down by many adhesions. During removal it burst, and a straw-colored fluid escaped. The left ovary was atrophied and dragged far up on the left side, and ensheathed by firm adhesions.

Dr. Jones also presented some microscopical specimens, showing the uterine appendages in a case of

UTERINE MYOMA.

The right tube, measuring ten inches in length, was coiled upon itself and filled with bloody serum. The closed fimbriated extremity was four and three-fourths inches in circumference.

The muscles of the walls of the tube had almost entirely disappeared, being replaced by fibrous connective tissue. A few remnants of the muscle-fibres had become waxy, and these were breaking down into inflammatory corpuscles. The epithelia of the mucosa had entirely disappeared, no remnants of them were left; and there were only a few remains of the folds of the mucosa left, and these were entirely changed to fibrous connective tissue, almost cicatricial. There were many newly formed blood-vessels with thick walls, and the newly formed fibrous connective tissue was crowded with inflammatory corpuscles.

The left tube was five inches in length and filled with pus. Almost the entire muscular structure of this tube also had disappeared, or was changed to fibrous connective tissue, and this new tissue was crowded with inflammatory corpuscles which, in many places, formed abscesses. The few scattered bundles of smooth muscle left were in a state of intense inflammation and beginning ulceration.

Also, in the left tube, the epithelia of the mucosa were entirely destroyed, and the mucosa was crowded with in-

flammatory corpuscles. Only a few blunt remnants of the folds of the mucosa were left, and these were in a state of intense inflammation. Amid these thickly crowded inflammatory corpuscles were seen in every part coarse bundles of fibrous connective tissue, showing that the mucosa had previously been invaded by a plastic inflammation, of which, probably, there had been repeated attacks. Also in the mucosa, or in the previously formed tissue, there were seen many newly formed blood-vessels.

The ovaries also gave evidences of extensive changes from long-existing disease; only a vestige of normal ovarian structure was seen, nor could an ovum be found in either ovary. Though the pathological changes showed long-existing chronic disease, yet portions of the ovary were in a state of acute inflammation; thus it would be properly diagnosed

SUBACUTE OÖPHORITIS.

When it was remembered that the disease of the appendages had been existing a long time, most probably antedating the formation of the myoma, Dr. Jones thought that the question might fairly be raised whether the diseased appendages had not produced the myoma.

Dr. H. M. Biggs presented a number of specimens illustrating a series of aneurisms. The first was one of

ANEURISM OF THE ARCH OF THE AORTA,

situated at the junction of the transverse with the descending portion, removed from the body of a negro dying in the penitentiary. The man presented signs of lobar pneumonia, but had no symptoms suggesting an aneurism. He was suddenly taken with syncope while at stool, and died in a few minutes. At the autopsy an aneurism of the arch of the aorta was found, which had caused erosion of the vertebræ, and had ruptured into the œsophagus. The lesion in the left lung was a very striking one, and its exact nature had not yet been ascertained. There was a very distinct and sharply defined red and white mottling, which at first sight seemed to be caused by multiple lympho-sarcomata. The microscopical examination had not been completed, but it had gone far enough, however, to show that this was not the lesion.[1]

The President said that the gross appearances in the lung were similar to those seen in the lungs of children

[1] At a meeting of the Society, held on May 23d, Dr. Biggs reported that he had completed the microscopical examination of the lung, and had found that the peculiar mottling was caused by a combined lobar and broncho-pneumonia. The red color was due to the lobar pneumonia, and the white areas showing through the red were the infiltrated walls of the bronchi.

who had died of lobular pneumonia, especially when the disease occurred secondarily to diphtheria.

The second specimen was one of

DISSECTING ANEURISM OF THE AORTA.

In this, the starting-point for the separation of the coats of the aorta was in its first part, just above the valves, and the dissection had proceeded up over the arch, and then down, involving the entire length of the thoracic aorta, passing through the diaphragm, and terminating in the abdomen. The man had died suddenly. No history of the case was obtainable.

Dr. Biggs also presented a third specimen showing

MULTIPLE ANEURISM OF THE AORTA.

First, there was a dilatation of the aorta just above the valves. Then, upon the transverse portion of the arch, immediately before its termination in the descending portion, there was a saccular dilatation, and from this arose a second sac. Finally, there was another aneurism on the descending aorta. The woman from whom the specimen was taken was an inmate of the workhouse, and had died of pneumonia. During life a small pulsating tumor had been noticed, but there were no other signs indicative of the condition found after death.

Dr. H. P. Loomis presented a specimen of

ABSCESS OF THE LIVER, WITH PERFORATION OF THE DIAPHRAGM,

sudden death occurring under ether. The patient, a man about thirty-eight years of age, came under observation on March 2d. His family history was good. He acknowledged having had syphilis, and said he was accustomed to go on periodical sprees. He had enjoyed perfect health up to four years ago, at which time, during a period of eight months, while residing in Kansas City, he had severe attacks of malarial fever, accompanied by diarrhœa and bloody stools. After leaving Kansas City he recovered his accustomed health until a year ago, when, after taking cold, he was seized with an attack of dysentery. The patient gradually recovered from his dysentery, but found now that any indiscretion in eating or in the use of stimulants would bring back a diarrhœa, accompanied by tenesmus and the passage of blood. Last Christmas the patient went on a spree, and after that he was much worse, having on an average as many as seven bloody movements daily, accompanied by tenesmus. Lately he complained at times of intense pain over the region of the liver, and was slightly jaundiced.

On examination all the organs were found in apparently healthy condition, with the exception of the liver, which was enlarged, the left lobe projecting about two inches below the free border of the ribs. The organ was tender on pressure. The patient's general condition improved during March, although the dysenteric movements continued, and percussion showed that the liver was increasing in size. He had no fever nor chills at any time. On April 2d the patient began to grow worse, the signs of subacute pleurisy on the right side developed, and the dysenteric movements increased up to about twelve a day. On April 6th the fluid in the right pleural cavity had increased to such an extent that an aspirating needle was introduced at the level of the sixth rib, two and a half inches from the median line, and twenty ounces of amber-colored serum, containing some flocculi of lymph, were withdrawn.

A few days after, when the needle of a hypodermic syringe was introduced one inch below and one and a half inch to the outer side of the point where the aspirating needle was previously introduced, a reddish purulent fluid was withdrawn, which, on microscopical examination, presented the appearance of fluid from an abscess of the liver. About two inches above the point whence the purulent fluid was withdrawn, a needle was introduced and clear serum was obtained. The case was diagnosed "an abscess of the liver," and an exploratory opening was advised.

On April 14th the patient was placed on the operating table and ether administered. Suddenly he became cyanosed and stopped breathing. All means to restore him failed, and he died without a cut being made.

Autopsy, made thirty-six hours after death: The right pleural cavity, for its upper three-fourths, was filled with serum in which were shreds of fibrin. At the lower fourth was a pus-cavity, enclosed by adhesions, which communicated, by an opening in the diaphragm close to the ribs, with an abscess of the liver. The opening in the diaphragm would barely admit the index-finger.

The abscess of the liver was a small superficial abscess, situated in the back portion of the right lobe, four and a half by four inches in diameter, and about two inches in depth. The weight of the liver was six pounds, and its structure was normal. The left lung was normal. The right lung was compressed against the vertebral column by the fluid in the right pleural cavity.

The heart weighed 250 grammes, and showed marked dilatation of the left ventricle, with thinning of the walls. The thickest part of the right ventricle was hardly half an inch thick. There was no valvular lesion, but quite extensive atheroma of the aorta and of the coronary arteries.

A microscopical examination of the cardiac muscle showed very extensive interstitial myocarditis.

The other organs were normal, with the exception of eight or ten dysenteric ulcers involving the cæcum.

The two interesting points of the case were: 1st, the peculiar encysted condition of the pus from the abscess after it had perforated the diaphragm, together with the serum in the upper portion of the pleural cavity; 2d, the sudden death from only a few whiffs of the ether, due, no doubt, to the extensive myocarditis which was not recognized during life, although the patient had been examined by some of the best diagnosticians in the city.

The President reported the case of a young man who had a history of gall-stones and an affection of the liver. He was expectorating a very foul-smelling, purulent fluid. Paracentesis was performed without ether, as it was feared that the other lung might become infected by the passage into it of this purulent matter during narcosis. The chest being opened, a sound was passed down through the diaphragm into the liver. The patient was at death's door for three days, but then began to rally, and is now a strong and healthy man.

Dr. H. J. Boldt presented a specimen of

EXTRA-UTERINE PREGNANCY.

The patient was referred to him, on account of a tumor in the abdomen, by Dr. Krollpfeiffer. She had recently passed through an attack of acute peritonitis, and, at the time Dr. Boldt first saw her, had a temperature of 102° F. in the axilla, and presented the appearance of one suffering from pyæmia. The tumor extended as high as the umbilicus, and the first thought was that it was a gravid uterus, especially as the menses had stopped seven months before. Examination, however, showed that the uterus was empty, and that the tumor lay to the left of this organ. The case was then diagnosticated to be one of abdominal pregnancy, and an operation was advised and agreed to by the patient. The operation was one of unusual difficulty, as the intestines were very strongly adherent to the sac. The right tube could be traced, but the corresponding ovary could nowhere be found. The walls of the sac were very thick, and an attempt was made to suture them to the abdominal wound; but this was only partially successful. The patient did well for about twenty-four hours, but then began to sink, and died in a few days. The fœtus had died at about the sixth month.

Dr. Frank Ferguson presented a specimen showing an

ANEURISM OF THE ASCENDING PORTION OF THE ARCH OF THE AORTA OPENING INTO THE PULMONARY ARTERY.

The specimen was taken from the body of a man, forty-five years of age, who was admitted to St. Luke's Hospital, in the service of Dr. F. P. Kinnicutt, on February 17, 1888.

He entered the hospital at 5 P.M., suffering from extreme dyspnœa. His face and hands were cyanotic. The heart, action was very rapid, and there were murmurs which were masked by the presence of numerous moist sounds over the chest during respiration. He could not lie down, and suffered intensely from dyspnœa. He became more and more cyanotic, his pulse was imperceptible at the wrist, and after a slight exertion he suddenly died.

The *autopsy* was made twelve hours after death. The heart was found increased in size; the ventricles were dilated, and there was fat in the muscular tissue. The aortic and mitral valves were competent. There was an aneurism as large as an ordinary hen's egg in the anterior wall of the ascending portion of the aortic arch. There was found a rupture of the wall of the sac and an erosion of the posterior wall of the pulmonary artery, establishing a free communication between the aorta and the pulmonary artery by a circular orifice one-half inch in diameter. This orifice was situated just above the pulmonary valve. One of the cusps of the pulmonary valve was adherent to the wall of the artery for a distance of one-fourth of an inch, which must have rendered the valve incompetent.

A second specimen was also one of

ANEURISM OF THE ARCH OF THE AORTA,

taken from the body of a man, fifty-three years of age, who was admitted to the New York Hospital, in the service of Dr. E. L. Partridge, on April 5, 1888. Nine months previous to his admission he had malarial fever, and eight months later he had a slight attack of rheumatism.

For the past few months he had had muscæ volitantes, headache, dyspnœa on exertion, slight palpitation, morning nausea, and vomiting. During the three weeks preceding his admission into the hospital he passed a diminished quantity of urine of high color, but showed no other nephritic symptoms.

He had a well-marked alcoholic habit. For some years he had been subject to a slight cough, which, during the five months preceding his residence in the hospital, was accompanied by a whitish expectoration. During the last

three or four weeks of this time his abdomen became distended, the distention increasing his dyspnœa. On admission his appetite was poor, and his bowels were constipated, but his chief complaints were weakness, cough, distention of the abdomen, and dyspnœa.

Percussion revealed dulness from the second rib, on the left side, to the left apex, but below the second rib the percussion note was normal. The expiration was prolonged and low-pitched, and there were sonorous rhonchi on inspiration, and on expiration many sibilant râles and sonorous rhonchi, with an occasional coarse, moist râle.

Posteriorly, over both sides, the physical signs were similar, but more marked in degree. The superficial cardiac area could not be determined, owing to pulmonary resonance. The aortic sounds were normal. At the apex the cardiac sounds could not be heard. The pulse was small and wiry, and the legs were markedly œdematous.

His case was diagnosticated as chronic diffuse nephritis and cirrhosis of the liver. Under appropriate treatment he improved considerably until April 15th, when he felt pain in his left chest, his respiration became gasping, and he suddenly died. The autopsy revealed 3,000 c.c. of pure blood in the left pleural cavity. There was an aneurism of the transverse portion of the arch of the aorta, ovoidal in shape, 14 ctm. in length and 10 ctm. in diameter. The sac was almost filled with laminated fibrine. The orifice of communication with the aorta was oval in shape, and 5 by 4 ctm. in size. The sac had ruptured on the left side, giving rise to the escape of the blood into the left pleural cavity by a slit-like opening, 2 ctm. in length. The aorta was very atheromatous and dilated. The great vessels arising from the arch were to the left of the opening in the aortic wall, and the blood in the aorta entered freely into these vessels. The left subclavian vein contained a small thrombus. The valves of the heart seemed competent, and the heart was normal in size, while the muscular tissue was anæmic. The kidneys were found in a state of chronic diffuse nephritis. There was much fibrous tissue in the liver ; the lungs were normally supplied with blood and well distended with air, and they covered in the aneurism completely. The heart was displaced downward slightly. The œsophagus and trachea were apparently free from pressure.

Dr. Ferguson then presented another specimen showing an

ANEURISM OF THE THORACIC AORTA OPENING INTO THE ŒSOPHAGUS.

It was taken from the body of a man, fifty years of age, who was admitted to the New York Hospital, under the

care of Dr. George L. Peabody, on September 22, 1887. Three years previous to admission he began to have pain in the left shoulder, which gradually grew worse, and one year later he developed a cough, accompanied by a white expectoration. Ten days before his admission he suffered from dyspnœa and dysphagia. His appetite was poor, and the bowels were constipated, but his pain was his chief complaint.

The cardiac area was found diminished in extent, and the cardiac sounds were faint, but otherwise normal. The pulse was of fair tension, and the arteries were tortuous and atheromatous. He was poorly nourished. There was no œdema. On the back, occupying the inter-scapular region, and extending to the left side, there was a large tumor measuring twelve inches in vertical diameter and eight inches horizontally. Over this swelling expansile pulsation was felt, but no murmur could be heard. Over the entire extent of the swelling there was flatness. During his residence in the hospital he suffered considerable pain, which was always, however, relieved by morphine. About ten minutes before he died he suffered intensely from dyspnœa, and had also a severe attack of coughing and vomiting of blood.

At the autopsy there was found a large aneurism, occupying the posterior half of the left pleural cavity, above the seventh rib, and extending four inches beyond the median line on the right side.

The aneurism was filled with laminated fibrine and partially coagulated blood. When the sac was removed, with a portion of the vertebral column, it was found to measure nine inches transversely and seven and one-half vertically. The third and the seventh ribs were partially eroded, and those between them completely severed. The sac communicated with the aorta by an opening one inch in diameter, and located three inches below the origin of the left subclavian artery. On the median line, in the posterior wall of the œsophagus, on a level with the bifurcation of the trachea, was a small opening through which blood escaped from the aneurismal sac. This opening was one-fourth of an inch in diameter. Several of the dorsal vertebræ were very deeply eroded. The heart was normal in size, and the valves were competent.

Dr. J. West Roosevelt presented two specimens of

SARCOMA OF THE MEDIASTINUM.

Case I.—Anne S——, aged thirty-eight years, was admitted to Roosevelt Hospital on July 26, 1883. Mother died of phthisis. Seems to have had no important sickness till last March. Then had "pleurisy" lasting three

weeks. About April 15th returned to work. Soon had dyspnœa on exertion, cough in the morning, with whitish sputa; occasional pain in back. Six weeks ago a swelling appeared in the right arm and wrist, and soon spread up the arm and involved the shoulders, head, and neck. During the last few weeks the patient had suffered from darting præcordial pains. The urine was acid, sp. gr. 1.027, contained a trace of albumin, but no casts—there were abundant urates. Diagnosis: Signs of pressure on the trachea or bronchi, most on right side. May have aneurism pressing on cava descendens and trachea. Œdema due to thrombosis. Both radials are very feeble, right possibly a little more than the left, but no marked difference. Sibilant rhonchus takes the place of the respiratory murmur on the right side in front, on the left side front exaggerated respiratory murmur. Marked dulness in the right chest in front and behind. Absent vocal fremitus on right side. No heart murmurs.

Several severe attacks of dyspnœa, without spasm of glottis, occurred on August 14th, and the following day the patient died.

Autopsy, August 16, 1883.—The heart was of normal size. Ante-mortem clot in right ventricle and auricle, and running into pulmonary artery. In the thorax was a new growth occupying the entire mediastinal space, surrounding the main bronchi of the right lung, causing almost complete obliteration of the superior vena cava, in which was a firm thrombus. The tumor extended upward in front of the trachea as far as the thyroid, sending projections upon each side of the larynx. It was adherent to the last cervical and first dorsal vertebra, and extended forward to the first rib, to which it adhered on the right side. It extended downward to about an inch above the diaphragm. A large part seemed to be composed of enlarged and adherent lymphatic glands. The tumor on section was white and pinkish-white in different places. Some portions contained a good deal of "cancer juice" and some were quite dry. The jugular, innominate, and subclavian veins on the right side all contained thrombi, where they entered the tumor. The walls of the jugular on the right were completely infiltrated, and the lumen of the vessel was almost obliterated.

The tumor, under the microscope, was found to be formed of a fibrous tissue stroma, which had embedded in it large and small round cells. It was very vascular. The lymphatic glands in the neighborhood were infiltrated with similar growth. The walls of the bronchi and of the large veins near the tumor were similarly infiltrated.

Case II.—William H——, aged eleven years, admitted August 25, 1883. The patient was moribund on admis-

sion. Child had been sick for only two months. He had œdema of the face and a cough. About six weeks ago a swelling as large as a goose egg appeared in the axilla. It was not painful and soon disappeared. Dyspnœa had been present for six weeks, worse at night. The child had lost flesh, especially during the last two weeks. On admission there was marked orthopnœa and considerable œdema of the upper extremities was noted. The patient died the day following admission.

Autopsy.—On opening the thorax a new growth was seen occupying the entire mediastinum, sending a process up along the left of the trachea as far as the larynx. It compressed the trachea to about one-half its normal size, and compressed and almost obliterated the descending cava and right jugular and innominate veins, also the right carotid and subclavian arteries, and slightly the transverse arch of the aorta. The left carotid and subclavian veins were also compressed. The left jugular and innominate and subclavian veins could not be found. The growth infiltrated the walls of these vessels. The bronchi of the left lung, for a distance of about an inch and a half from their entrance into the lung, were infiltrated and compressed. The upper lobe of the left lung was in great part converted into tumor-tissue, with a few masses of compressed lung-substance between. Numerous patches of gray lobular pneumonia were seen in the remaining portion of this lung. The upper lobe adhered throughout to the chest-wall. The left chest was nearly full of clear serum, but there was none in the right. Pericardium was distended with liquid blood. The valves, as well as the myocardium, seemed normal. The tumor resembled the other in microscopic structures.

Tumors of the mediastinum are rather rare, and they are of interest on account of this comparative rarity. In the first case the diagnosis became pretty clear. The physical signs indicating pressure on a large bronchus, and the symptoms pointing to the obstructed superior cava, were very pronounced. From the histological structure of the neoplasms it seems probable that they had their origin in the bronchial or mediastinal glands.

The Society then went into executive session.

Stated Meeting, May 23, 1888.

W. P. Northrup, M.D., President, in the Chair.

Dr. Biggs reported from the Committee on Microscopy that the lung showing a red and white mottling, presented at the meeting of April 25th, was the seat of a combined lobar and broncho-pneumonia, the red coloration being due to the lobar pneumonia, and the white showing through being the infiltrated walls of the bronchi.

The President presented, on behalf of candidates, two specimens, one showing a fracture of the ulna, and the other, abscesses of the mammæ in a child.

The President also presented, for Dr. Frank W. Hopkins, of Fishkill, N. Y., specimens showing

TUBERCULOSIS OF THE LYMPHATICS, ULCERS OF THE COLON, AND CROUPOUS COLITIS AND NEPHRITIS.

A female child, eight and a half years of age, had conjunctivitis, was listless and anæmic, and apparently quite sick. For a time she improved, but about three months before death she began to have elevation of temperature, and occasional chills and sweating. For more than a month before death she began to emaciate, albumin and casts appeared in the urine, and diarrhœa developed. For a time the treatment seemed to be of some avail, and her general condition improved, but her temperature again became elevated, there was tenderness over the spleen, enlargement of the liver, with moderate jaundice. No albumin was present at this time, but abundant hyaline and granular casts. Continuing in this condition, she gradually failed and died.

On autopsy numerous submaxillary and cervical glands were found to be enlarged, cheesy, and diffluent in their centres. The bronchial glands were moderately but uniformly enlarged, and all cheesy. There were no tubercles in the lungs, and no consolidation; only bronchitis and moderate congestion. The mesenteric and retroperitoneal glands were uniformly enlarged and cheesy, though no discrete tubercles could be found in the adjacent tissues, peritoneum, or diaphragm.

The kidneys were the seat of chronic nephritis. One was larger than the other. The markings were indistinct, the capsule adherent, and the cortex showed cicatrices. No tubercles were found within or upon either kidney.

The most marked lesion of the viscera was within the

colon. At the cæcum, and about eight inches from it, upon the ascending portion of the colon, were two large ulcers extending entirely round the inner surface of the gut, and two and a half inches in breadth. When laid open, the wall seemed to have been ulcerated over nearly a square area. The lower or cæcal ulcer was limited on its upper margin by a narrow linear groove running transversely around the gut. The second large ulcer was limited both above and below by a similar narrow groove or sharp linear ulcer. These limiting grooves were large enough to allow an ordinary lead-pencil to lie in them. The edge toward the main ulcer was deeply undermined and overhanging and ragged, while the edge facing outward was clean cut. As for the main ulcers, they were honey-combed. A probe passed easily in a winding course beneath the undermined mucous membrane.

These two tubercular ulcers consisted, then, of undermined mucous membrane, limited on the margins by linear ulcers having one—the inner—edge overhanging, and the outer sharply cut, or punched out. The remaining mucous membrane of the colon was the seat of croupous inflammation, having upon its swollen and congested surface a shreddy layer of fibrine. The jejunum and ileum were the seat of catarrhal inflammation. Liver and spleen showed no tubercles, and the other viscera were normal.

Dr. H. M. Biggs presented a specimen of

PRIMARY TUBERCULOSIS OF THE UTERUS AND TUBES.

The clinical history was imperfect. The patient had had hectic fever and was supposed to be phthisical. At the autopsy the uterus was found to be about the size of an orange, and to contain considerable cheesy pus. Its walls were about the thickness of the walls of the bladder. Both the uterus and the tubes exhibited well-marked lesions of advanced tuberculosis, and there were tubercular ulcers in the vagina. There was also present recent tubercular inflammation of the peritoneum and pleura.

A second specimen was one of

THORACIC ANEURISM,

which was especially interesting because of its large size and from the absence of symptoms directing attention to its presence during life. The patient was a helper in Charity Hospital and had died suddenly. He had had no symptoms pointing to heart trouble. At the autopsy a large aneurism was found filling the whole of the mediastinum and a large part of the pleural cavity. It contained a clot, six inches in diameter, composed of laminated fibrine. The aneurism was not ruptured, and the cause of death was not clear.

A third specimen was one of

DERMOID CYST OF THE KIDNEY.

One kidney was of large size and normal in appearance; the other was small and contained six or seven cysts varying from the size of a pea to that of a filbert. The cysts contained fatty, pasty matter, composed of cholesterin crystals, fat-globules, epithelium, and compound granular corpuscles.

Dr. Biggs also presented a fourth specimen, of

MULTIPLE SOFTENING OF THE BRAIN,

which was remarkable on account of the large number and extent of the spots of softening. The brain was from a man who had been demented, but had always been able to be around. He was found one morning unconscious in bed, and so remained until he died. At the autopsy two large cysts were found in the frontal lobe on the left side, and one in the right frontal lobe. In the parietal and occipital lobes the convolutions were atrophied, and numerous small spots of softening—ten or twelve in all—were found scattered throughout the convolutions.

Dr. Boldt, referring to the first spe imen presented, asked what symptoms were present, and what reason the speaker had for supposing that the tubercular process was primary in the uterine appendages.

Dr. Biggs said that there had been no symptoms of uterine disease. He had assumed that the tuberculosis was primary in the tubes rather than in the uterus, as that was more commonly the case. Primary tuberculosis in the uterus, extending thence to the tubes, was a very rare condition. The lesions in the tubes and uterus gave proofs of being so much older than those in the peritoneum and pleura that it was evident that the process had begun in the former.

The President stated that the specimen of ulcerative colitis, presented for Dr. Hopkins, had been preserved for six weeks in a solution recently prepared by Dr. Prudden for preserving specimens in gross. It had been found to be very satisfactory in its action. The solution would be presented and its composition described at the next meeting of the Society.

Dr. Francis Delafield then showed with the lantern a series of photo-micrographs illustrating the lesions of

ACUTE BRIGHT'S DISEASE.

He said: "I have been invited to demonstrate to you this evening some micro-photographs and drawings which illustrate the lesions of acute Bright's disease. But before

doing this I must occupy a little time in telling you what it is that I wish to demonstrate.

"In considering the lesions of any part of the body there is a manifest advantage if we are not obliged to look upon them as morbid changes peculiar to that part of the body, but rather as changes of the same kind that we find elsewhere. This we can do as regards the lesions of acute Bright's disease.

"In many parts of the body we find what is called acute exudative inflammation—a morbid process in which the active share is taken by the blood-vessels. The serum, fibrine, and pus-cells which belong to this inflammation are derived from the contents of the blood-vessels, while changes in the tissues themselves are absent or of secondary importance.

"One of the ordinary forms of acute Bright's disease is such an exudative inflammation of the kidneys,

ACUTE EXUDATIVE NEPHRITIS.

Such a nephritis occurs as an idiopathic inflammation, and as a complication of scarlatina, diphtheria, and many of the infectious diseases. The products of inflammation, albumen, casts, puscells, and blood are regularly present in the urine. The excretory function of the kidneys is more or less interfered with; there is fever and prostration; there may be added anæmia, cerebral symptoms, and disturbances of the circulation.

"In the simplest examples of this form of nephritis we find, after death, no changes in the kidneys. The inflammatory products have all escaped with the urine, and no evidences of the inflammation are left. More frequently more or less of the inflammatory products remain in the kidneys, and we find, after death, cast matter, coagulated matter, and white and red blood-cells in the tubes, and white blood-cells and serum between the tubes.

"Still further than this there may be changes in the renal epithelium and in the glomeruli. The epithelial cells may be swollen or broken down, or detached from the walls of the tubes. The cells which line the capsules of the glomeruli are swollen, those which cover the capillaries and are situated within them are increased in number and size. Such a nephritis is essentially a transitory lesion. If the patient does not die from the immediate effects of the disease, the kidneys return to their natural condition.

"Secondly, we find that persons who have taken poisons such as mercury, arsenic, or phosphorus, or who have suffered from the infectious diseases, regularly exhibit changes in the cells of the viscera. These cells are simply swollen, or are infiltrated with fat, or die. How far such

changes are degenerative or inflammatory is not yet definitely determined.

"In these ways are produced the second form of acute Bright's disease,

ACUTE PARENCHYMATOUS INFLAMMATION OR DEGENERATION OF THE KIDNEY.

Such a lesion is always secondary to one of the causes just mentioned. The urine is unchanged, there are no renal constitutional symptoms. But if the changes in the renal epithelium are decided and rapidly developed there will be added changes in the blood-vessels of the kidneys, albumen and casts make their appearance in the urine, and the quantity of the urine is diminished. After death we find the renal epithelium swollen, or fatty, or necrotic. In some cases the vessels are congested, and there is cast matter in the tubes.

"Thirdly, we find in different parts of the body inflammation, with the production of new connective tissue, and such inflammations are apt to persist. So we find as the third form of acute Bright's disease a condition which we may call

ACUTE DIFFUSE NEPHRITIS.

Such a nephritis is often idiopathic, it complicates scarlatina, diphtheria, and pregnancy. The urine with such a nephritis contains large quantities of albumen and casts. The patients exhibit anæmia, dropsy, cerebral symptoms, and marked disturbances of their general health. After death we find in the kidneys the same changes as are found in exudative nephritis, but, in addition, a growth of new connective tissue and of the capsule cells of the glomeruli. The growth of new connective tissue begins in the cortex and follows the lines of the larger vessels. Its arrangement is often in the form of regular wedges. The growth of the capsule cells is often enormous, so that the tufts are compressed and finally atrophied.

"The lesions are permanent lesions, and when they have once begun their natural tendency is to continue until a large part of the kidney is replaced by new connective tissue, and a large number of the glomeruli are atrophied."

The speaker then made a lantern demonstration of the changes in the kidneys which he had described.

On motion of Dr. J. C. Peters, a vote of thanks was passed to Dr. Delafield for his interesting demonstration.

The President referred to the cases, not infrequently met with in autopsies upon children, whose urine had contained albumen and casts, where at post-mortem absolutely

nothing abnormal was to be detected upon the closest examination in the kidneys.

These cases seemed to be most satisfactorily explained by the classification described under acute exudative nephritis in which the lesion is of the blood-vessels, and the exudative products are washed away in the urine.

The Society then went into executive session.

Stated Meeting, June 13, 1888.

W. P. Northrup, M.D., President, in the Chair.

Dr. G. C. Freeborn stated that Dr. J. M. DaCosta, the Middleton Goldsmith lecturer for the present year, had declined to receive any compensation for his lecture and had requested the Society to apply the amount ($100) to any purpose which might seem desirable. The matter had been referred to the trustees, and they had decided that the most fitting tribute to Dr. DaCosta's generosity would be to purchase some microscopes for the use of the Society, and had requested the speaker to procure them. He had done so, and now exhibited them. He had succeeded in getting two microscopes, one having an Abbé condenser a $\frac{1}{3}$ and a $\frac{1}{5}$ objective, the other having no condenser and being of a low power. The microscopes would be engraved with an inscription to the effect that they were the gift of Dr. DaCosta.

Dr. J. J. Griffiths presented a specimen of

ACUTE PHTHISIS IN A YOUNG CHILD,

with very extensive deposits of tubercle in the lungs and lymphatic system.

M. ——, aged thirteen months, a foundling, was presented at the Out-door Department of the New York Foundling Asylum, on April 22, 1888, with a history of severe indigestion and diarrhœa of three days' standing. The disease yielded to treatment, and the child remained well until May 17th, on which date she was returned with motor paralysis of the entire left side, including the face. The pupil of the same side was dilated; no paralysis of sensation; moderate diarrhœa; otorrhœa, left side; temperature, 100° F. Six days later it was noted that the left hemiplegia continued, and in addition there was loss of power of the right leg. During the next ten days motion slowly returned to the left arm and leg. The child was returned to hospital on May 30th, and the subsequent history was kindly furnished to the speaker by Dr. Lynde, the house-physician.

Upon admission the cervical, epitrochlear, and inguinal glands were enlarged, there was ptosis of the left lid, and the child refused to nurse.

On June 8th the child was much worse; respiration rapid and labored. There was dulness on percussion anteriorly and posteriorly on the right side, and the respiratory sounds were absent over the whole lung. No

retraction of the intercostal spaces; diminution in movement of the chest-wall. The left side was tympanitic on percussion, and bronchial breathing and coarse bronchial râles were heard; marked retraction of the intercostal spaces; temperature, 104° F.

During the four days following, her condition remained unchanged, and she died in collapse, June 12th.

Autopsy, two hours after death. Child fairly well nourished. The brain was markedly anæmic, but otherwise normal. There was a small amount of bloody serum in the pleural cavity. On the right side the superior portion was collapsed and the inferior portion entirely occupied by a large tuberculous deposit, which at several points had broken down into small cavities. Costal pleura normal; pulmonary pleura thickly studded with miliary tubercles. On the left side there were abundant pleuritic adhesions over the anterior and lateral surfaces of the lung, binding it closely to the chest-wall.

In the substance of the entire lung were many deposits of yellow, caseous, tuberculous material, varying in size and consistency. The lymphatic system was largely involved. The cervical, bronchial, retro-peritoneal, inguinal, and epitrochlear glands were all much enlarged and caseous, and many were diffluent at their centres. The liver was anæmic and fatty. The other organs were normal.

The case was interesting chiefly for these reasons: 1. Because of the absence of cerebral lesions to account for the marked hemiplegia which existed; 2, that so extensive a deposit should have been confined entirely to the lungs and lymphatic system, no tubercle having been found in any other part of the body; and, 3, that in this instance epitrochlear enlargement was due to tubercular and not syphilitic disease.

The President wished to emphasize especially two points of interest in this case. It had been dogmatically asserted that enlarged epitrochlear glands were always an evidence of constitutional syphilis, but here no signs of that disease were discoverable. The second point was the presence of symptoms pointing to cerebral disease, although no brain lesions were found post mortem.

Dr. Ridlon thought it conceivable that there was an hereditary syphilitic taint in this case. He had seen a good many children, with a clear hereditary history of syphilis, who had tubercular bone lesions, and he had thought that there might possibly be some connection between hereditary syphilis and tubercular disease in children.

Dr. Biggs had never seen anything exactly like this specimen. The lungs contained large tubercular masses with diffuse miliary lesions.

Dr. Myers presented a

PLACENTA

with the following history of the labor and of the autopsy made on the child :

The mother was admitted to the Foundling Asylum in labor. She had œdema of the legs, and examination of the urine showed a specific gravity of 1.024, acid reaction, granular and epithelial casts, and enough albumin to cause it to solidify on boiling. The quantity passed in twenty-four hours was but four or five ounces.

The labor was rapid, but the head rested at the vulva, and the abdomen caused delay when presenting. The cord was about the neck, but parted on slight traction without hemorrhage.

The placenta was friable and pale, portions of it coming away like cotton-wool when pulled upon, and it was thought to be œdematous.

The child had œdema of the legs, arms, and face, and ascites. It weighed four and one-eighth pounds. Punctate hemorrhages were seen in the skin, especially in that of the legs. The infant was thought to be seven months old. It breathed twelve or fifteen times with assistance, and lived fifteen minutes.

Autopsy.—The abdomen contained eleven and one-half ounces of dark-brownish fluid.

The foramen ovale and ductus arteriosus were open, but the heart was otherwise normal ; its weight was three and one-half drachms.

The liver and spleen were somewhat enlarged, and the former contained a number of chalky masses the size of a millet-seed.

There were numerous punctate hemorrhages seen upon the surface of the kidneys and in section of the organs. The tibia was decalcified, and showed normal epiphyseal junctions.

The microscopical examination of the placenta and of the liver of the infant had been made by Dr. Northrup, who reported as follows :

"The chalky, millet-seed-sized masses in the liver were found to be carbonate of lime. The tissue, when hardened and decalcified, showed them to be situated in the veins.

PHLEBOLITHS.

According to Delafield and Prudden 'the changes which occur in a thrombus after its formation may be either in the direction of degeneration or organization. In some cases they seem to undergo a simple shrinkage and decolorization, and to become infiltrated with the salts of lime, forming the so-called *vein-stones* or *phleboliths*' ("Handb. of Path. Anat. and Hist.").

"The capillaries were seen to be filled with white blood-cells, distinctly stained in the microscope specimen herewith shown. The capillaries were dilated and the nearest liver-cells were atrophied. About many veins and phleboliths there was a moderate increase of organized connective tissue.

"Placenta: The shreddy tissue making up the bulk of the body, when removed with care, was found to be a gnarl of fibres. These represented the normal stroma and were expected to tell something of the nature of the process. The stroma was slightly thicker than normal, but well-organized, and the vessels with it were normal. The thickening meant diffuse moderate placentitis. The long bones were examined for the condition of the epiphyseal junctions. The tibia was decalcified and sections for the microscope prepared from the upper epiphysis. It was normal.

"The condition of the placenta was not explained by the microscope findings except negatively. There was no evidence of syphilis, and the moderate placentitis would not explain it. The mother was œdematous; the child was in a condition of general anasarca, and in my opinion the placenta was likewise œdematous."

Dr. W. H. Porter thought that there was general dropsy of the whole placental tissue, to which was to be attributed its macerated and atrophic condition. He had seen a similar placenta in a case in which the mother was perfectly healthy, but all the organs of the child were waxy. The cord was œdematous, and the œdema of the placenta resulted in its separation, and in the loss of the child at the seventh month.

Dr. Prudden said that a placenta, resembling this one exactly, had been sent recently to the pathological laboratory of the College of Physicians and Surgeons. No history of the labor had accompanied the specimen. The child was apparently six or seven months old, and had no œdema. Either this condition was not so rare as one would be led to suppose, from the scanty reference to it in the text-books, or else there was a remarkable coincidence in the report of these three cases.

The President did not believe that such cases were at all common. He had examined a great number of abnormal placentæ, but had never before seen one like this. He asked whether there was any objection to an explanation of the condition on the theory of universal œdema of mother, child, and placenta.

Dr. H. M. Biggs presented a specimen showing chronic

CARDIAC THROMBOSIS.

The patient was admitted to Charity Hospital suffering from advanced valvular disease of the heart, having dysp-

nœa, general anasarca, and rapid and irregular heart action. Under the administration of infusion of digitalis and Hoffman's anodyne he improved for a few days, but then sudden heart-failure occurred and he died.

Autopsy.—There was general œdema, all the large cavities being also filled with fluid. There were evidences of recent pericarditis. The heart was large, weighing about twenty ounces. The mitral valve was the seat of old endocarditis, some calcification being present, and was narrowed. The left auricle was enormously dilated, and contained in its upper part a thrombotic deposit one and a half inch in thickness, the central portion of which had undergone puriform softening, but had not yet opened into the auricle. The right auricle contained a number of small thrombi, varying from the size of a hickory-nut to that of a large walnut. The pulmonary artery was much dilated, and there were numerous points of hemorrhagic infarction in the lungs.

The President showed a piece of intestine which had been placed during warm weather in a quart bottle containing a new preserving fluid compounded by Dr. Prudden. The intestine had been in the fluid for nine weeks, but was well preserved, even showing distinctly the different colors of the fresh specimen.

Dr. T. M. Prudden presented the formula for the

NEW PRESERVING FLUID

referred to by the President.

Water	1,000	c.c.
Common salt	100	gm.
Saltpetre	25	"
Carbolic acid	5	"
Glycerine	15	c.c.
Amylic alcohol	50	"
(or Ethylic alcohol	100	")

Specimens should be first soaked in a strong brine and then placed in a large quantity of this fluid. He believed the mixture would be found to serve a useful purpose in the temporary preservation of gross pathological specimens without changing their color or otherwise altering their general appearance. The mucous membranes were particularly well preserved in the fluid.

Dr. J. Lewis Smith reported a case of

SEPSIS IN AN INFANT.

The child was born in the Foundling Asylum on May 13th. The mother did well, having no febrile disturbance. Before the separation of the cord it was noticed that the child's umbilicus was sore and discharging a little pus.

The speaker first saw the umbilical phlegmon when the child was seven days old. At this time three whitish patches, about half the size of a silver dime, were noticed on the hard palate near the velum. There was also purulent conjunctivitis in one eye, notwithstanding the fact that Credé's method of instillation of argentic nitrate solution is always employed in the institution. The faucial trouble increased, and the phlegmon also continued to extend, until the child died at the age of fourteen days. The autopsy was made by Dr. W. P. Northrup, who reported as follows: "The abdominal wall, for the distance of an inch in every direction from the umbilicus, was swollen, the central portion being brown and dry. The peritoneal surface was congested and was adherent to the omentum at the level of the umbilicus. From this point a leash of dilated vessels, an inch in width and between three and four inches long, passed to the transverse colon. The umbilical and hypogastric arteries contained clots. Points of hemorrhagic pneumonia were scattered through all the lobes of the lungs. The stomach showed the lesions of acute gastritis; the mucous membrane was intensely congested, and there was an abundance of tenacious mucus. The kidneys were soft, flabby, and dusky in color. The liver and spleen were normal.

"The anatomical diagnosis was

UMBILICAL PHLEGMON,

peritonitis, acute gastritis, and hemorrhagic pneumonia."

The microscopical and biological examination was made by Dr. T. Mitchell Prudden, who reported as follows:

"The small ragged cavity at the umbilicus contained a moderate amount of pus and of cell-detritus, and enormous numbers of bacteria, of various forms, the spheroidal form predominating. The tissues of the abdominal wall about the umbilicus were infiltrated with fluid, fibrine, and pus. Scattered about in this exudation mass were small spheroidal bacteria. The hypogastric arteries and the umbilical vein were plugged with a clot for from one-half to three-quarters of an inch from their origins. Their walls were greatly thickened by infiltration with inflammatory exudate. Both in the lumina of these vessels along the sides of the clot and in the lymph-spaces in their walls were enormous numbers of small spheroidal bacteria. These bacteria were present in the umbilical vein beyond the limits of the clot, in the direction of the liver.

"The kidneys showed moderate parenchymatous degeneration. The consolidated areas in the lungs were

due to a nearly complete filling of the air-spaces and smaller bronchi with blood.

"Cultures made from the inflamed tissue about the umbilicus, and from the edges of the sloughing cavity, showed several species of bacteria common in the air and in the fæces of children. In addition to these, the staphyloccus pyogenes aureus was present in large numbers.

"A set of cultures from the inside of the umbilical vein at a little distance from the sloughing cavity, revealed the presence of staphylococcus pyogenes aureus and streptococcus pyogenes, together with other forms.

"Cultures from the liver showed large numbers of staphylococcus pyogenes aureus, with considerable numbers of a stout bacillus similar to the one abundant in the sloughing cavity.

"From the consolidated regions in the long tissue enormous numbers of bacilli developed in a nearly pure cultuie, which corresponded in its biological characters to the bacterium lactis aërogenes of Escherich."

Dr. Smith then spoke of the treatment employed, and referred to the disinfection of the room, after a case of contagious disease, by means of burning sulphur. He read a letter from Dr. E. R. Squibb, of Brooklyn, in which the writer stated that

BURNING SULPHUR IS OF DOUBTFUL EFFICACY

as a disinfectant, the weight of the highest authorities in bacteriology being against it, notwithstanding that it is still largely used by very intelligent bodies in large institutions, boards of health, etc., where it would not be likely long to maintain an unearned confidence. Its efficacy would doubtless be greatly increased were the fumes allowed to mingle with the vapor of water discharged at the same time in the apartment. Many bacteriologists have admitted that burning sulphur would kill bacteria, but not their spores.

Dr. Prudden said that the bacteriological examination would seem to show that the child had been the victim of infection with the ordinary "suppurative bacteria," and with the bacteria of fæces. Many of these fæces bacteria were found in the lungs of the infant. As regards the restraining influence of iodoform upon the growth of bacteria, he had mixed bacteria and iodoform together very thoroughly, and had found that the bacteria grew well in cultures made from this mixture.

The Society then went into executive session.

Stated Meeting, June 27, 1888.

FRANK FERGUSON, M.D., VICE-PRESIDENT, IN THE CHAIR.

DR. J. J. GRIFFITHS presented a specimen, on behalf of a candidate, showing the engagement of the omentum in the lateral perforations of a glass drainage-tube.

DR. H. J. BOLDT presented a specimen of

ABSCESS OF THE UTERUS, PUERPERAL PYO-SALPINX, AND INTRA-PERITONEAL ABSCESS.

The patient, J. G——, aged twenty-two, married for five years, had been referred to him by Dr. L. Weiss. She had had three children and no miscarriages. The last child had been born four weeks before the speaker saw the case. The labor was normal. Two hours after delivery the woman had a chill, which was followed by a high temperature and profuse sweating. The axillary temperature varied from 103° to 105° F. There was constant pain over the lower portion of the abdomen, the uterus was large and flabby, and on either side of it was felt a fluctuating movable mass, supposed to be the distended Fallopian tube. The case was thought to be one of septic salpingitis following delivery, a diagnosis which was strengthened by the facies of the patient.

Abdominal section was decided upon, but the patient died in collapse a few hours later. The operation was very difficult on account of the very dense adhesions which had partly been diagnosed before operation. On the right side the tumor felt proved to be the distended Fallopian tube, which ruptured while separating the adhesions, discharging the purulent contents into the abdomen. On the left side, however, the tumor which was thought to be the distended tube proved itself to be an encapsulated intra-peritoneal abscess, which also burst during the operation; the Fallopian tube, although inflamed, and in consequence considerably enlarged, presented nothing unusual. The ovary was very intimately attached to the uterus, so that removal would have been impossible without injury to the uterus itself. Drainage was used.

At the autopsy the remaining tube was found firmly occluded about three-fourths of an inch from its uterine extremity. In cutting through the anterior uterine wall, an abscess was found in the substance of the muscular structure, which contained about two drachms of pus. The cavity of the abscess had the shape of a round canal,

traversing nearly the whole breadth of the uterus, and situated in the upper half of the body; it was entirely shut off from connection with any other structure, and was regarded as the result of an infarction by the speaker. The diameter of the abscess canal was three eighths of an inch. The cavity of the uterus measured three and one-half inches. The large size and flabby condition of the uterus, the speaker regarded as partly due to the abscess in the muscularis.

Dr. Ferguson had recently seen an abscess in the wall of the large intestine, occurring as the result of occlusion of the artery by an embolus. The abscess had ruptured into the lumen of the gut, but this was probably the result of manipulation post mortem. The case was one of malignant endocarditis.

Dr. H. P. Loomis presented a specimen of

PATENT FORAMEN OVALE,

in a man fifty-three years of age. The man had always been troubled with shortness of breath and cyanosis. He said that his mother had told him that he was a "blue baby." He had lived in Switzerland until ten years ago, when he came to this country. Since coming here he had been much worse and had been unable to work. Physical examination showed a dilated heart with an indistinct murmur, which was later resolved into a mitral regurgitant.

At the autopsy there was found an infarction in the lower lobe of each lung, one of which was old, the other recent. The heart weighed a pound and a half, was flabby, dilated, and had very thin walls. The interauricular septum was thinned, and at its lower portion was a perforation admitting a quill, which was, however, closed by a valvular flap of tissue which seemed to be competent to prevent the passage of blood. The kidneys presented a classical picture of infarctions, some comparatively recent, others older.

Dr. Loomis also presented other specimens showing

MASTOID DISEASE, PACHYMENINGITIS, AND MULTIPLE VISCERAL ABSCESSES.

Owing to the patient's limited command of English it was impossible to obtain a satisfactory clinical history. It was, however, learned that he had suffered for about a week from intense hemicrania accompanied with a discharge of pus from one ear. Soon after the pain ceased the left arm and leg became cold, and then gangrene appeared in the hand and foot.

At the autopsy the mastoid cells were opened and were found to contain a few drops of thick pus. There was

pachymeningitis on the same side, and suppurative thrombi were seen in the lateral sinus. The axillary artery was occluded at its point of junction with the brachial, and there was suppuration along the course of the arteries. Pyæmic abscesses were found in the lungs and spleen.

Dr. Ferguson, referring to the first specimen presented, said that he had seen a number of cases of patent foramen ovale in adults in which there had been no heart symptoms during life. In every instance there was a flap of tissue present serving as a valve. He thought the condition of the heart-walls, which were thin, flabby, and fatty, was sufficient to account for the later symptoms of dyspnœa and cyanosis in this case.

Dr. Ferguson then exhibited a microscopical slide of a section through the mucosa of the stomach of a man who had died of pernicious fever, showing the

MALARIAL PIGMENT.

The vessels were filled with the pigment, looking as if they had been artificially injected. The man had been brought to the hospital in a partially comatose condition, and never rallied sufficiently to give a clear account of his illness. All that could be learned was that he had had a bilious attack for three days, and was found on the street in a nearly unconscious state. He gradually grew worse, the temperature toward the end being 106°, and died three days after admission. The diagnosis was made *intra vitam* by the examination of the blood. A large amount of pigment was found in all the organs, but especially in the brain and stomach.

The Society then went into executive session.

Stated Meeting, October 10, 1888.

W. P. NORTHRUP, M.D., PRESIDENT, IN THE CHAIR.

DR. T. H. MYERS presented, on behalf of a candidate, a *small vesical calculus*, which was discovered by the cystoscope, dislodged, and subsequently passed per urethram.

DR. T. MITCHELL PRUDDEN presented two candidate-specimens, the first showing some pieces of *iron wire perforating the stomach of a pig*, the second being one of *multiple abscesses of the liver.*

DR. HODENPYLE presented some microscopical specimens prepared according to

WEIGERT'S CORRECTED METHOD FOR STAINING FIBRIN IN SECTIONS.

The method, which is described in detail in the *Fortschritte der Medicin,* vol. xviii., p. 693, 1888, is as follows: The sections are spread out on glass slides, dried with filter paper, stained for five minutes in gentian anilin water, and again dried with filter paper. They are then placed in Lugol's solution of iodine for from thirty to sixty seconds, and finally decolorized in a mixture of zylol one part, and anilin-oil two parts. They are mounted in balsam. The specimens should he hardened in alcohol. By this method the shreds of fibrin are stained a deep blue, all the other parts being decolorized. They show much better in the daytime than by artificial light.

DR. MARY A. DIXON JONES presented a series of seven specimens showing that

MISPLACEMENTS OF THE UTERUS

are in many instances due to and complicated by diseases of the uterine appendages.

All the patients gave a history of long suffering and sterility, and had been through the usual course of treatment with pessaries and other supports, but without relief. Operation disclosed the uselessness of such forms of treatment, as the pelvic organs were displaced and bound down by inflammatory exudates, and extensive disease of the ovaries and tubes coexisted. In some instances the ovaries were atrophied, in others enlarged, and in still others they were the seat of purulent degeneration. Pyosalpinx was also of frequent occurrence.

Laparotomy was successfully performed in all seven cases, and the patients have been free of pain since.

Dr. Van Santvoord presented a specimen of

RUPTURE OF THE ARCH OF THE AORTA.

The patient was a man, forty years of age, who was admitted to hospital for some cerebral trouble, of which no definite history could be obtained. It was thought that the affection was syphilitic in its origin, and the man was put upon appropriate treatment. Eight hours before death the patient became markedly cyanotic, and a murmur was heard which was supposed to indicate aortic trouble. At the autopsy the aortic valves were found to be unaffected. There was, however, an aneurism of the arch of the aorta, situated just above the valves, which had ruptured into the superior vena cava. The rent was about one-third of an inch in length. The cerebral lesion was found to consist in multiple spots of softening from vascular disease.

Dr. Van Gieson exhibited two slides showing specimens of

ACCESSORY PANCREAS IN THE WALL OF THE DUODENUM, SIMULATING A TUMOR.

One of the specimens formed a whitish nodule, about 8 x 4 mm. in diameter, situated in the musculosa of the duodenum of a new-born child, and projecting in the lumen of the gut. The nodule was composed of lobuli of the pancreas lying between the mucosa and serosa.

The second specimen was taken from a man aged thirty-five. There was a white flattened mass, 1½ ctm. by 6 mm. in diameter, in the wall of the duodenum, through which the common bile-duct passed. The mass consisted of lobuli of the pancreas, lying between interlacing bands of the musculosa.

Dr. Ferguson had seen a similar case some time ago in St. Luke's Hospital. The supernumerary organ was about half an inch in thickness, and it was identical in structure with the true pancreas. The speaker had neglected to note whether there was an excretory duct present.

Dr. Prudden said that these cases were interesting in that there was danger of mistaking the glands for carcinomatous tumors, as they bore a very close resemblance, in gross appearance, to the latter when the sections were made in a certain direction. In the specimens presented there was no difficulty in recognizing the ducts as ducts, but if they had been cut in another direction the resemblance to that form of carcinoma which looks like adenoma would have been very striking.

The President said that he had been interested in the specimens for the very reason mentioned by Dr. Prudden

for he had seen one of them before, and at the first glance had pronounced it carcinoma.

Dr. H. M. Biggs presented a specimen showing

RUPTURE OF THE HEART,

which had occurred in a man seventy-four years of age. He was an inmate of the almshouse, and was working in the kitchen one day when he suddenly fell down, and died before the physician could reach him. At the autopsy it was found that a rupture of the anterior wall of the left ventricle had taken place. The tear was a ragged one, 6 or 7 mm. in length. The pericardium was distended with blood.

A second specimen presented was one removed from the body of a girl, showing

PRIMARY TUBERCULOSIS OF THE KIDNEY.

The other organs were free from disease, except the lungs, in which some recent miliary tubercles were found. Tuberculosis of the genito-urinary organs is not very uncommon, but it was rare, in the speaker's experience, to find it localized in the kidney.

Dr. Biggs also presented a specimen of

MULTIPLE GALL-STONES IN THE LIVER,

removed from the body of a man who had died in Bellevue Hospital. No history of the case had been obtainable. At the autopsy were found evidences of extensive fibro-serous peritonitis; there were also pleural adhesions and acute pericarditis. All through the liver the smaller bile-ducts were dilated, some forming cysts of considerable size and containing gall-stones. Small abscesses had formed on the upper surface of the liver and there were adhesions between this viscus and the diaphragm. There was one abscess in the left lobe, 3 ctm. in diameter, which, like the cysts, contained gall-stones.

There was also a smaller abscess in the right lobe. From one of these abscesses the inflammatory process had extended into the pericardium, and from the other into the pleural cavity. The gall-bladder contained no calculi, and the larger ducts were also free from them.

Dr. Prudden said that he had never seen a case in which the gall-stones in the liver were so numerous.

Dr. L. Emmett Holt presented the lungs of a child, nineteen months old, an inmate of the New York Infant Asylum, which showed

BRONCHO-PNEUMONIA AND PULMONARY ABSCESS COMPLICATING ULCERATIVE COLITIS.

The child was taken, on August 11th, with acute diarrhœal symptoms and high temperature. The fever subsided

after four or five days, but the diarrhœa persisted, with variable severity. The movements were from four to six daily and varied in color from yellow to yellowish green and black, with much mucus but no blood. At times they were quite offensive. On September 11th the temperature again rose rapidly, reaching 104.8° F. It continued elevated until death, fluctuating between 100° and 103° F. The signs of commencing broncho-pneumonia were found at this time, first at the left base behind, then extending to the right base, and finally along the whole spine on the left side. No exacerbation of the diarrhœa accompanied these symptoms, the movements continuing about as above throughout the illness. The spleen was markedly enlarged, so that it could be felt an inch and a half below the ribs. The child emaciated rapidly, was exceedingly irritable, and it was only by the greatest patience that any physical examinations of the chest were made. She died, October 8th, having lost six pounds during the two months of her illness.

Autopsy, twenty hours after death. Brain: Pia œdematous; otherwise normal. Lungs: Adhesions over middle and lower lobes, mostly old and firm; similar adhesions over the whole left lung, behind and laterally. The breaking up of the adhesions opens, at the apex of the left lung, an abcess with ragged walls, containing about an ounce of thick, creamy pus, not fetid. Pus welled up in the trachea upon pressure, but it seemed rather doubtful whether a free communication with a bronchus had existed during life, as no pus to speak of was expectorated. The posterior part of the rest of the upper lobe and the whole lower lobe showed the usual changes of acute broncho-pneumonia. The same appearances were seen in the right lower lobe, posterior half, and a few scattered spots elsewhere. No small abscesses were present; bronchial glands much enlarged, not cheesy. Heart pale. Liver: Enlarged and quite fatty; weight, one pound two ounces. Spleen: Firm and adherent to abdominal wall, and about four times the usual size; weight one ounce six drachms. Kidneys: Enlarged; weight, one ounce two drachms; very marked cloudy swelling. Stomach: Contained large firm curds, and showed small ecchymoses in mucous membrane, near pyloric end; otherwise, no gross changes. Intestine: Small intestine pale throughout; has a "washed out" appearance; villi distinct; Peyer's patches normal; intense congestion in the rectum and sigmoid flexure but only moderate in the rest of the large intestine; rectum markedly thickened; solitary follicles not much enlarged. In the transverse and descending colon and rectum are many irregular ulcers, the largest half an inch broad; for

the most part they involve the mucous membrane only. In the transverse colon some go to the circular muscular coat. They are all old in appearance. Mesenteric glands were enlarged and pale; not cheesy. The association of broncho-pneumonia with entero-colitis was something very commonly met with. A process in the lung, such as shown in the specimen, was decidedly uncommon. It suggested, however, some possible connection between it and the intestinal lesion.

The ground had been taken by some that all the cases of broncho-pneumonia complicating intestinal disease were due to infection.

GENERAL MILIARY TUBERCULOSIS.

Dr. Holt also presented specimens from an infant thirteen weeks old, illustrating general tuberculosis with obscure symptoms. The child was premature, mother delicate, and grandfather tubercular. On admission in July the child weighed four pounds; two days before death its weight was five pounds six ounces. The mother had but little milk, and this deficiency was made up by malted milk.

Except a very large spleen and dyspeptic symptoms, as shown by occasional vomiting and abnormal stools, the child gave no evidence of disease beyond the fact that it did not thrive. There was no cough. During the last five days the temperature was kept at, and it never rose above, normal. It was occasionally subnormal, and this condition existed for twenty-four hours before death. The autopsy was made thirty-six hours after death.

The brain showed a few miliary tubercles, but no evidences of meningitis.

The lungs were closely studded with miliary tubercles throughout, and showed many minute cheesy nodules, but no large ones; no pleurisy, and not much broncho-pneumonia. Bronchial glands were enlarged, cheesy, and broken down.

The spleen was five times normal size, miliary tubercles were seen on its surface, and the whole organ was thickly studded with small cheesy nodules.

The mesenteric glands were large and cheesy, slight catarrhal changes were found throughout the intestinal tract, and in the ileum two or three minute ulcers, not connected with Peyer's patches, which were normal.

The case was of interest as showing how few symptoms even a wide-spread tubercular process often gives in a very young infant, and, secondly, in view of the great enlargement of the spleen. This symptom had been advanced by some recent writers as of a good deal of value in making a diagnosis of tuberculosis in infancy. The

speaker, however, had found it a very untrustworthy symptom.

Dr. Van Santvoord said that in his experience the diagnosis of tuberculosis in young children was very difficult. The symptoms were very irregular, and there was often nothing upon which to base a certain diagnosis.

The President thought that there was no rule as to the relation between enlargement of the spleen and tuberculosis.

On motion, the lung shown by Dr. Holt was referred for examination to the Committee on Microscopy.

Dr. Frank Ferguson presented a specimen, showing the lesions of

ANGINA LUDOVICI,

removed from the body of a man thirty years of age, a native of Italy, married, and a laborer by occupation. He was admitted into New York Hospital on September 1, 1888. For five days previous to his admission he had noticed a swelling in the floor of his mouth, and a gradual stiffening of his jaws. He was, on admission, unable to move the inferior maxilla, and suffered great pain. He could give no cause for the swelling, but says it commenced opposite the angle of the lower jaw. He had much swelling and induration in the floor of the mouth, extending upon the outer side of the inferior maxilla as high as the alveolar margin. The anterior surface of the neck was also swollen and indurated down as far as the cricoid cartilage. There was distinct fluctuation beneath the floor of the mouth on the left side. Dark-colored offensive pus was drawn from this region by the aspirating needle. The mucous membrane was raised from the alveolar margin of the inferior maxilla, along the incisor and canine teeth behind, and as far back as the first molar on the left side. On the day of his admission an incision was made two and one-half inches in length, and running obliquely downward and backward from the hyoid bone to the inferior maxilla. This incision was carried through skin and superficial subcutaneous tissue, and opened into a cavity filled with several ounces of offensive pus. The cavity was irrigated daily with a solution of bichloride, 1 to 5,000, drainage-tubes inserted, and a bichloride dressing applied. On the night following his admission he had a chill, and complained of a difficulty in breathing and flatulency. He was very restless during this night. During the second, third, and fourth days in the hospital he perspired very profusely, his pulse was very rapid and weak, and his temperature rose from 101° F. to 104° F. On the fourth day he continued restless, frequently attempting to get out of bed. He was constantly and care-

fully stimulated, but he gradually sank and died, having a temperature just before death of 103° F.

At the autopsy there was found a pint of purulent fluid in the pericardium. Both layers of the pericardium were covered with recent lymph. There were numerous gangrenous cavities under the root of the tongue, which communicated with the wound along the lower border of the jaw, and from which pus extended downward through the mediastinum to the pericardium. The pus infiltrated the planes of fibrous tissue along the trachea, œsophagus, and great vessels in the neck. The other organs were normal. The fluid in the pericardium, as well as sections of the necrotic tissue beneath the tongue and lymph covering cardiac surface, were stained for micro-organism, and large colonies of micrococci were everywhere observed. They were the same as those ordinarily found in erysipelas.

Dr. Ferguson also presented a second specimen, showing

ACUTE MILIARY TUBERCULOSIS,

removed from the body of a boy, thirteen years of age, who was admitted into the New York Hospital on September 27, 1888. He gave no morbid family history, and had no bad habits. It was learned from his mother that he had been treated in St. Luke's Hospital, five years previous to his admission into this hospital, for hip-joint disease, and was discharged cured. At the same time he suffered from a fractured patella, which also healed. He was in his usual health until two weeks before his admission, when he commenced to complain of pain in his left knee, varying with pain in left hip, for which no cause could be assigned; there was no swelling, heat, nor redness of the knee. About one week later he began to complain of a very severe general headache. At this time the pain seemed to leave his knee. He vomited, but not frequently or severely, his bowels were fairly regular, and there were no ocular symptoms. He had no chills, but suffered from high fever, with maximum elevation of temperature toward night. He had recently suffered from burning pain in his eyes, dryness of mouth and throat, and great thirst. He had a slight cough. He had epistaxis on two different occasions. His appetite was poor. On his admission he had no nausea nor vomiting, but his bowels were constipated. He complained of no abdominal pain or tenderness. He was very weak and suffered much from headache. A harsh, respiratory murmur was audible over both lungs, and the respiration was prolonged and accompanied by an occasional subcrepitant râle. His urine was alkaline, of amber color, specific gravity,

1.025, and had a trace of albumin, and microscopic examination showed some pus and blood-cells. On October 2d he commenced to develop a delirium of a low, muttering type; later, cephalalgia and subsultus tendinum, with well-marked restlessness; and later on he became entirely unconscious, having irregular convulsive movements of the muscles of the body, especially marked in the muscles of the arms and forearms. The pupils were both markedly dilated, the tongue was dry and brown in the centre, the abdomen was slightly distended and tympanitic, and the abdominal veins were enlarged and very prominent. There was some resistance to motion of the neck, but there was no pain nor tenderness. He vomited twice on the night of October 3d, and he had conjugate deviation of the eyes to the right, and his head was also turned to the same side. On the afternoon of the third he was quieter, sinking into a comatose condition, with the left side of the body motionless and apparently paralyzed. On the fourth he was perfectly comatose, the eyes were partially closed, but there was no strabismus and the pupils responded to light. On the sixth he seemed much improved, and was apparently conscious, making attempts to respond to directions given him, and he executed voluntary movements of the right side, but still showed much lethargy. The pulse had been getting gradually more rapid and feeble, but he remained in much the same condition as on the day before. There was a slight ulceration on the right cornea. On the sixth the coma gradually deepened, the temperature rose, the respiration became deep, labored, and rapid, and he continued to fail until 9.30 P.M., when he died. The temperature ranged from normal to 104° F., the pulse from normal to 160, and the respiration from 20 to 44 per minute. The urine, toward the close, had to be drawn with the catheter; it contained a trace of albumin, and responded to Ehrlich's test. It contained hyaline casts and a small quantity of pus. The bowels were persistently constipated. Ice was applied to the head, strophanthus and whiskey were given, and the patient was fed on peptonized milk.

Autopsy.—On inspection the body was found extremely emaciated. The rigor mortis was fairly well marked. The left leg was found two inches shorter than the right, and everted. The left hip-joint was anchylosed. There was a sinus in the upper third of the thigh, on its outer aspect, leading to the joint. The convolutions of the brain over the vertex were slightly flattened, and there was slight œdema of the pia mater. There was pus in the meshes of the pia mater covering the interpeduncular space, and the pia mater in the Sylvian fissures was

studded with tubercles. The lateral ventricles were slightly dilated and filled with pale fluid. The floors of the ventricles were softened, as were also the corpus callosum and septum lucidum. On the right side there were a few young tubercles found in the ascending frontal convolution. The lungs, kidneys, spleen, and small intestines contained numerous small miliary tubercles, and there were very small tubercular ulcers in the ileum.

Dr. T. Mitchell Prudden presented a specimen of

CIRRHOSIS OF THE LIVER IN A RABBIT,

showing small nodular areas scattered throughout the liver-substance. There was nothing in the nature of the experiments to which the animal had been subjected to explain the occurrence of cirrhosis.

The Society then went into executive session.

Stated Meeting, October 24, 1888.

W. P. Northrup, M.D., President, in the Chair.

Dr. T. Mitchell Prudden presented a uterus showing

EXTENSIVE LACERATION OF THE CERVIX.

The case was one of protracted labor with transverse presentation. Version was performed, but death resulted from septicæmia. The history was as follows:

"A woman thirty-five years of age, who had borne three children, and who had chronic phthisis involving both upper lobes, was in the eighth month of pregnancy, had been feeble and in bed for some time, and finally became delirious. This is all that could be learned of her previous history. On her admission to the hospital she was mildly delirious; had a temperature of 100° F.; was feeble; the urine was scanty, with fifteen per cent. albumin and granular casts. The fœtal heart-beats were heard, but there were no symptoms calling more detailed attention to her pregnant condition.

"On the day after admission she, for the first time, complained of abdominal pain. Examination revealed a transverse presentation with the hand and arm protruding from the cervix. On the introduction of the fingers for the performance of version, three or four ounces of a black grumous material came from the uterus. Version and delivery were performed without difficulty, no unusual force being used. There was no hemorrhage—the child was dead; an intra-uterine douche of bichloride of mercury, 1–3,000, was used. Two hours after delivery the temperature rose to 102° F.; but the woman appeared brighter and her general condition improved. She was now passing not more than seven ounces of urine. On the following day her temperature was normal, and ranged from that to 101.5° F. Her pulse was better, but her general condition was worse. Under free stimulation she died, without further rise of temperature, about forty-eight hours after the delivery.

"*Autopsy.*—Brain and meninges, heart and spleen normal; lungs, chronic phthisis involving both upper lobes; liver, fatty; kidneys, small, rough, cortices thin and irregular; peritoneum normal, save that over the *cul-de-sac* of Douglas it was thickly bestrewn with small gray tubercles; uterus firmly contracted, its inner aspect posteriorly showing normal surface of placental attachment. The cervical portion was deeply and extensively lacerated, its surface rough and gangrenous. The uterine sinuses and both ovarian veins

were filled with dense, firm thrombi. The right ovarian vein was so extremely distended with the thrombus—which was in part white, and apparently of considerable age—as to measure nearly five-eighths of an inch in diameter. The bladder was normal; the urethra presented on the lower side a small whitish patch of croupous exudate.

"This case seems to me to be of interest because it affords an excellent example of the rapid overwhelming of the system with poisonous material from an injured surface, without the occurrence of any well-defined inflammatory lesion. How long in this case the labor may have been going on before its discovery is only a matter of conjecture, but it seems most likely that the laceration, and very probably the infection, had occurred before the admission of the patient to the hospital.

"I think that it is convenient, in the present state of our knowledge—although we have perhaps no strict scientific reasons for doing so—to group

THE PUERPERAL INFECTIONS

into two classes, one which we may call the *putrid infection*—perhaps most often caused by the putrefaction of retained placental fragments, or, as in our case, by gangrene following a more or less extensive injury; and that which is well enough termed *purulent infection* or puerperal pyæmia, in which there are somewhere foci of suppurative inflammation. The latter form of puerperal infection would be that which more strictly comes under the heading of puerperal fever.

"I have taken the liberty of calling attention to this grouping of these puerperal conditions, simply to emphasize that class of which this is so excellent an example, in which women die after delivery, and sometimes very soon after, without much rise of temperature, and without the existence of any appreciable inflammatory lesions. They not only die, but seem often to do so to the great surprise of the attending physician, who is prone to call upon the pathologist for the discovery of a cause for death. And this cause the pathologist has frequently to discover or to postulate without the aid of any well-defined anatomical data. It is perhaps only the bacteriologist who has seen much of the effects of pathogenic bacteria on animals, who is ready to give an unquestioning acquiescence to a "cause of death" which leaves as little trace of its modus operandi as does in many cases puerperal septicæmia."

Dr. George C. Freeborn presented a specimen of

EPITHELIOMA OF THE BODY OF THE UTERUS.

The patient, aged fifty-six years, has been married forty years. Two years ago she noticed a scanty discharge

from the uterus; this increased, became watery in character, sometimes bloody, and finally muco-purulent. She had complained of but little pain. The uterus was removed by hysterectomy.

The uterus measured 12 ctm. in length and 8 ctm. across the horns. The body was rather globular in form. On opening the uterus, an irregular roundish mass, measuring 6 × 5 ctm., was found growing from the posterior wall of the body. It projected about 1½ ctm. above the mucous membrane. The surface was soft, white in color, and had a "cauliflower" appearance. Microscopical examination showed the growth to be epithelioma.

Dr. Freeborn also presented a second specimen, showing

RUPTURE OF THE COLON FOLLOWING OVARIOTOMY.

The patient was operated on October 1st, both ovaries being removed, they being cystic. Several adhesions to the intestines were found with the left ovary, and were broken up. After breaking up a rather firm one, severe hemorrhage followed; the bleeding point was found and ligated, and the operation finished in the usual manner.

On October 4th symptoms of rupture of the intestine occurred. Owing to the weak condition of the patient, exploratory laparotomy was not deemed advisable. General peritonitis set in, and the patient died October 24th.

Post-mortem examination showed general purulent peritonitis, the abdominal cavity being filled with pus, and solid and liquid fæces. In the upper and left portion of the sigmoid flexure of the colon, an oval opening 2 inches long and ½ inch broad was found, the whole of the intestinal walls included in the above dimensions having sloughed away. At the upper end of the opening a silk ligature was found imbedded in the tissue. The rectum was very much contracted, probably from the want of use, as all fæcal matter had been discharged through the drainage-tube from the time of the symptoms of rupture.

The point of interest in this case is whether, in breaking up of the adhesion, the peritoneal and muscular coat was torn away, leaving only the mucous membrane. If this were the case, the latter, being deprived of its means of nourishment, would have sloughed, and thus caused the opening.

Dr. H. M. Biggs presented a series of specimens illustrating various forms of

DISEASE OF THE VERMIFORM APPENDIX.

The first specimen was accompanied by no clinical history, as the patient had died two or three hours after ad-

mission to the hospital. At the autopsy a general peritonitis was found. There was a perforation of the vermiform appendix, and a fæcal concretion was discovered in the abdominal cavity.

The second specimen was taken from the body of a patient suffering from typhoid fever, who had died with the usual symptoms of intestinal perforation. The autopsy revealed ulceration and perforation of both the ileum and vermiform. The ulceration in the latter was ragged and did not have the appearance of a typhoid lesion. If it were a typhoid ulcer, however, it would be a rather curious coincidence to have perforation of both the ileum and vermiform appendix.

The third specimen was one of

TUBERCULOSIS OF THE VERMIFORM APPENDIX.

About an inch of the extremity of the process was cut off from the remainder and was filled with a cheesy material. There was tubercular ulceration also in the ileum, but here there were no tubercles on the peritoneal surface of the gut, so that external inspection of the bowel would not have led the examiner to suspect tubercular ulceration.

Dr. Biggs then reported another case, of which the specimen was not shown, of impaction of fæcal matter in the appendix. The body was the size of the end of the little finger, and was situated at some distance from the opening. The opening was very much smaller than the fæcal mass, so that the latter must have been formed within the appendix, and could not have passed into it from the cæcum. These cases of vermiform perforation seem to be growing quite common, the speaker said; he had seen ten or twelve cases within the last two years.

Dr. Biggs then presented another specimen showing

MULTIPLE DIVERTICULA OF THE LARGE INTESTINE.

The pouches were small but quite numerous.

The last specimen was one of

RUPTURE OF THE UTERUS

occurring in a case of criminal abortion. The organ had been removed from the body of a girl sixteen years of age. The abortion had been produced at the third month of pregnancy. On the anterior wall of the uterus were to be seen several punctures, possibly made by a knitting-needle or a straightened hair-pin. In the lateral wall was a large laceration, an inch and an half in length, which had evidently been made by a different instrument, and probably at a later period than had been the smaller punctures.

The cervix was bruised, but was not involved in the laceration of the body of the organ.

Dr. Mendelson, referring to the specimens of vermiform disease, asked how much importance, in Dr. Biggs' opinion, was to be attached to the popular belief that the seeds of fruit were a cause of vermiform disease. Considering the great quantities of grape-seeds that are swallowed at this season, and the comparatively few cases of disease of the vermiform appendix, it hardly seems that the swallowing of seeds can be a very efficient cause.

Dr. Biggs said that he had never found any foreign bodies in the appendix other than fecal accumulations. He had, however, sometimes found other substances forming the nucleus around which the fecal matter had accumulated. He remembered one case in which the nucleus seemed to be a raisin stem. He did not believe that seeds had any influence in the production of vermiform disease.

Dr. John C. Peters said that one of the earliest specimens presented to the Society was brought by Dr. Alonzo Clark, in which there were several layers of strawberry-seeds in the vermiform appendix; these seemed to have been accumulating for several years, to judge from the appearance of the different layers. That they were really seeds, and not fecal accumulations resembling seeds, was proved by microscopical examination.

Dr. Prudden said that whether the seeds merely formed the nucleus, or were the whole accumulation, their presence was significant, and he asked Dr. Biggs whether he had often found foreign bodies as nuclei.

Dr. Biggs said that in the majority of cases that he had seen there were no seeds or other foreign bodies except fecal concretions. Out of a dozen or more cases that he had seen, however, during the past two years, there was but one in which a rupture of the vermiform had occurred, and no fecal concretion had been found in the abdominal cavity—aside from the one of ulceration complicating typhoid lesions in the ileum, which he had just reported. He believed that cases of simple necrotic ulceration of the vermiform appendix were not common, if they occurred at all.

Dr. B. F. Curtis referred to a case under the care of Dr. Abbe, in St. Luke's Hospital, in which a pin was found in the abdominal cavity. The case had been operated upon some months before and a sinus had remained. The pin was found at the second operation.

Dr. Prudden said that he had searched for seeds in all the cases which had come under his observation, but he had never yet succeeded in finding any. He had seen some bodies which looked like seeds, but examination

had always shown that they were not seeds, but simple concretions.

Dr. L. E. Holt had seen the case referred to by Dr. Curtis, at the time of the first operation, and he was quite positive that no pin was present at that time. As to the case of intestinal tuberculosis, he had also seen one in which there were no evidences of tuberculosis upon the peritoneal surface of the bowel.

Dr. Thacher said that he had recently made an autopsy in which he had found multiple diverticula of the intestine similar to those in the specimen shown by Dr. Biggs.

Dr. Mendelson recalled the case reported to the Society several months ago by Dr. Ferguson. He was present at the autopsy. A diagnosis had been made of abscess of the liver. The cause of the abscess was not evident at first, but it was traced at the autopsy. There was found in the mesentery, behind the descending colon, an abscess which, extending upward, involved the tail of the pancreas. In consequence, thrombosis of the pancreatic veins had occurred, the thrombotic process extending into the liver, and causing multiple abscesses there. The origin of the abscess behind the colon was not at first apparent, but on dissecting out the colon carefully, it was found to have many large and small diverticula, one of which having ulcerated, had given rise to all the trouble.

Dr. Biggs related a case of diverticulum of the ileum, in which a pin was found imbedded in the mucous membrane.

The President asked Dr. Biggs what was the nature of the instrument used in the case of criminal abortion reported by him.

Dr. Biggs replied that there had evidently been two instruments employed, for the punctures in the anterior wall were made probably by a knitting-needle or a straightened hair-pin. The lacerations in the lateral wall must have been caused by the use of a much larger and coarser tool. It was probable also, that the injuries had been inflicted at two different times, and that the first and more simple instrument having failed, more active measures were undertaken.

Dr. L. Emmett Holt presented the lungs of a child two years of age, showing

PRIMARY CROUPOUS PNEUMONIA, COMPLICATED BY PURULENT PLEURISY, AND TERMINATING IN GANGRENE.

Female child, aged two years, an inmate of the New York Infant Asylum. Stout and well until twelve days before death, when she was taken suddenly ill, with a tem-

perature of 102° F., which rose the same evening to 104°. From that time it continued steadily high, ranging between 103° and 105.5° F., on two occasions reaching 106°. It fell only once as low as 101.5°, on the day before death. With the temperature there were present all the usual symptoms of lobar pneumonia, and by the fourth day the physical signs revealed complete consolidation of the right upper lobe. On the sixth day friction-sounds were heard over this lobe, and the signs indicated an extension of the consolidation into the middle lobe in front and lower lobe behind. Up to this time there had been but little prostration. During the next two days there developed a marked change for the worse. The prostration was so great that the child was not disturbed for any further examination of the chest. In spite of very free stimulation she lost ground steadily, and died of exhaustion on the twelfth day. There were no cerebral symptoms present at any time.

Autopsy.—The brain was normal. The right chest contained six ounces of pus which was not fetid. This was contained in two cavities; the larger one embraced nearly the whole pleural cavity, and the smaller one was just beneath the sternum and contained only half an ounce. The visceral and parietal layers of the pleura were covered with a thick, shaggy layer of fibrin and pus.

The right lower lobe was compressed, but was otherwise normal. The whole upper lobe and the upper half of the middle lobe were hepatized, of a brownish-red color. On section there were seen in the upper lobe three or four gangrenous areas in the centre of the lobe, the largest being about three-fourths of an inch in diameter; they were broken down in the centre. The gangrenous odor was scarcely perceptible. The largest artery leading to this region of lung was plugged by a thrombus, white, firm, and adherent, and to all appearance ante-mortem in its formation. In the lower border of the upper lobe another wedge-shaped gangrenous spot existed, extending to the surface of the lung. A similar thrombus was found in a vessel here.

The left lung was somewhat emphysematous above, and congested below; there were a few minute spots of broncho-pneumonia in the posterior border of the upper lobe; otherwise this lung was normal.

The heart, pericardium, stomach, intestines, and spleen were normal. The liver was large and pale. The kidneys were one and one-half time the normal size, the cortex was pale and thickened, and there was the general appearance of quite a well-marked parenchymatous nephritis.

A microscopical examination made from the hepatized lung showed the exudation to be almost entirely fibrinous,

only a few small round cells and epithelial cells being seen in the alveoli.

Dr. Prudden said that, in the course of the examination with Dr. Northrup of a large number of cases of broncho-pneumonia in children, he had seen four cases, all in children not over three years of age, of genuine acute lobar pneumonia with extensive pleurisy. The gross and microscopical appearances of these lungs corresponded with those of acute lobar pneumonia. The question of these cases being genuine acute lobar pneumonia, similar to that so common in adults, was settled by the results of culture, which showed in all the cases the pneumococcus of Fraenkel in large numbers.

The President said that these four cases were only four cases out of a very large number, and that they were not cited as evidence of the comparative frequency of the disease in children, but of its rarity.

Dr. Thacher asked what these lungs looked like to the naked eye.

The President replied that they presented much the same appearance as the lungs of adults who had died from croupous pneumonia.

Dr. Van Gieson presented some sections of the spinal cord from a case of compression myelitis, showing

DEGENERATION OF THE ANTERO LATERAL ASCENDING COLUMN.

After hardening in Müller's fluid, a portion of the upper dorsal region, two and a half centimetres in length, was seen to be transversely flattened, the entirety of the gray matter was absent, and a considerable portion of the cord was softened. Above the softened portion the posterior median and direct cerebellar columns were lighter in color than the surrounding white matter. There was also a bilateral narrow, light-colored zone in the periphery, continuous with, and anterior to, the direct cerebellar column. Following the periphery of the cord, it extended forward to a point about opposite the middle of the anterior horn. Below the softened portion there were bilateral light-colored areas corresponding to the direct and crossed pyramidal tracts.

The Society then went into executive session.

Stated Meeting, November 14, 1888.

W. P. NORTHRUP, M.D., PRESIDENT, IN THE CHAIR.

DR. SELLEW presented some specimens illustrating the lesions of

GENERAL TUBERCULOSIS,

taken from a child three and a half years of age, who, up to within four days of her death, though convalescing from whooping-cough, had otherwise been well, with the exception of slight listlessness for about a week. Four days before death she was taken with vomiting and fever, followed by delirium, stupor, convulsive twitchings of the face, slight nystagmus, irregular breathing, and occasionally the "hydrocephalic" cry. Physical examination gave only negative results.

Autopsy.—Twenty-four hours after death. The body was well nourished. There was tubercular meningitis at the base of the brain gluing together the large fissures. Moderate exudation at the base. The convolutions were much flattened. The ventricles were enlarged and contained clear fluid. There were no tubercles found in the lungs. One or two bronchial glands were slightly enlarged and cheesy. The liver and spleen were normal, with the exception of the presence of small tubercles. The kidneys were slightly enlarged and the markings moderately distinct. There was moderate catarrh of the stomach and intestines, but no ulcers.

In connection with this case, it was thought to be of interest to ascertain the frequency with which the brain is involved in cases of acute general tuberculosis, and it was found, by reference to the history of cases at the New York Foundling Asylum, that of fifty-nine cases of general tuberculosis, thirty-nine were associated with tubercular meningitis, while in twenty there was no involvement of the brain. Thus, in about two-thirds of the cases there was meningitis, and in about one-third the tubercles were confined to the other viscera.

DR. LEWIS HALL SAYRE exhibited the head and neck of a femur, removed five weeks previously from a boy seven years of age, who was suffering from

HIP-JOINT DISEASE.

Up to the age of four years, the boy had enjoyed perfect health. At that time he had had a severe attack of diphtheria followed by serious inflammation of the eyes, which kept him confined to his room for some time. He also had severe middle-ear disease involving the mastoid

process. It was then that the first symptoms of hip disease manifested themselves. The course of the disease was marked throughout by comparatively little pain, although there had been suppuration and abscess formation. When the speaker first saw the boy the thigh was flexed at nearly a right angle with the trunk, and there was a large fluctuating tumor in the neighborhood of the trochanter major. It was decided to operate, both for the purpose of correcting the deformity and of giving exit to the pus. When the capsule of the joint was opened it was found that the head and neck of the femur were carious, and these parts were accordingly excised. The acetabulum was not diseased. At present the wound is completely closed, some motion at the hip is possible, and the boy bids well to make a fair recovery. Had it not been for the deformity which called for operation, this case, the speaker thought, would have done remarkably well without exsection.

Dr. Henry P. Loomis presented a stomach removed from the body of a man who had died from

ACUTE MERCURIAL POISONING.

The man, who was twenty-six years of age, took with suicidal intent ten grains of the bichloride of mercury in a glass of water. Soon afterward he repented of the act and applied for relief at the Chambers Street Hospital. His stomach was syphoned; after which eggs and milk were freely administered. He gradually became weaker, with increased frequency and great feebleness of the pulse, and coldness of the extremities. His pharynx was intensely congested, and he complained of severe pain on deglutition. There was constant pain in the epigastrium, and he complained of a sense of burning and constriction in the throat. He vomited constantly a greenish-colored mucus, occasionally streaked with blood, but at no time did the bleeding amount to a free hemorrhage. His urine was frequently examined and always found normal. The countenance assumed a turgid and anxious expression, and four days after taking the poison he died from exhaustion.

Autopsy.—Fourteen hours after death. The mucous membrane of the mouth, throat, and œsophagus was wrinkled and covered with a white coating. The stomach showed at the cardiac end, along the greater curvature, and especially near the pylorus, dark, mottled, and irregular patches; the rest of the mucous membrane, which appeared normal, was covered with a dark, thick, tenacious mucus. No blood was found in the stomach, but two superficial ulcerations were found near the pyloric valve. Microscopical examination of the irregular patches

showed that they were due to capillary hemorrhages in the mucosa. The intestines were normal, with the exception of spots of congestion and ecchymosis in the large intestines. The kidneys were apparently normal, but microscopical examination showed a slight amount of parenchymatous degeneration. The liver was intensely congested, but otherwise normal.

Dr. Loomis also presented a specimen of

ANEURISM OF THE ABDOMINAL AORTA,

from a man fifty-three years of age, who was admitted to Bellevue Hospital in a dying condition. No history was obtainable. The diagnosis of lobar pneumonia was made, and the presence of an aneurism was not suspected. The patient died twenty hours after admittance.

Autopsy.—Thirty-six hours after death. The lower lobe of the right lung was in the second stage of pneumonia. The heart was dilated. There was no valvular change. The thoracic aorta showed throughout its whole extent the evidences of a chronic endarteritis, calcareous plates being scattered in places along its wall. Communicating with the abdominal aorta, just below the cœliac axis, was a sacculated aneurism about the size of a small orange, projecting backward and to the left. The opening into the aneurismal sac measured about an inch across. The sac contained no fibrin or blood; along its lower wall was a calcareous plate half an inch square. The aneurism had given rise to no symptoms during life. The liver was cirrhotic, and the kidneys showed the changes of chronic diffuse nephritis.

Dr. Mendelson asked what seemed to be the actual cause of death in the case of mercurial poisoning.

Dr. Loomis replied that the real cause of death was obscure; the man simply grew weaker and weaker until he died.

Dr. Herman M. Biggs presented a liver removed from the body of a Chinaman who had died in Charity Hospital, which showed the presence in great numbers of

DISTOMA HEPATICUM.

The autopsy had been made only a short time before the meeting, and consequently he had not had time to prepare a full report of the case.

Dr. Biggs also presented a specimen showing

RUPTURE OF THE AORTA.

The patient was a woman, an inmate of the almshouse, who, while sitting quietly, suddenly fell over and died. At the autopsy there was found a rupture of the aorta immediately above the valve. At the seat of rupture the

adventitia was found dissected from the media for a short distance, but it was not properly a dissecting aneurism, for the blood had evidently made its way between the media and adventitia at the time the rupture occurred. At the point of rupture the aorta did not seem, to the naked eye, to be diseased, but was apparently unusually thin. There was atheroma, however, in the neighborhood. These cases of rupture of the aorta without previous dilatation were rare.

Another specimen illustrated

ANEURISM OF THE AORTA.

There was a large aneurism of the ascending portion of the arch of the aorta, with dilatation of the arch, and adhesions to the sternum. The man had been a helper in the hospital for some time. He had had no symptoms pointing to aneurism, and the condition was discovered only accidentally. The man died suddenly while at stool. There was no rupture of the aneurism, so that the immediate cause of death was somewhat obscure. Possibly death had resulted from suffocation caused by additional compression of the trachea during the act of straining at stool, as there was marked compression and flattening of the trachea from pressure. The aneurism communicated with the aorta through a large opening. The case occurred in the service of Dr. Janeway.

Another specimen presented was one of

RUPTURE OF AN AORTIC ANEURISM.

The aneurism occupied the ascending portion of the arch of the aorta, and the perforation was the size of a pin-hole. Death did not follow until the expiration of about three-quarters of an hour after the first symptoms of rupture occurred. This specimen was interesting for this reason, as death after rupture of aortic aneurism was usually instantaneous. The lesions found in the aorta were those of acute endarteritis.

The speaker said that he wished to call attention again to the frequency of aortic aneurism as a cause of sudden death. About six months ago he had reported thirty-four cases of thoracic aneurism with autopsies, and had laid especial emphasis upon the frequency of this affection as a cause of sudden death without previous marked symptoms. He thought that its frequency was not appreciated by the profession generally, because these cases did not often come to the autopsy table for examination. They were all coroner's cases, and if there were no suspicious circumstances attending death, the coroner's physician did not usually perform an autopsy, but ascribed death to cerebral hemorrhage or heart disease. In a

large proportion of such cases an autopsy revealed an aortic aneurism with or without rupture, or a rupture of the aorta or heart. He had presented two other such cases before this autumn, and the three cases now presented had come under observation during the preceding two weeks. The thirty-four cases presented last spring had, with two or three exceptions, been seen within a a period of about eighteen months. Aside from this point which he wished to emphasize, these cases had some features of especial interest. Many of the cases that he had reported had been seen through the courtesy of Dr. W. T. Jenkins, coroner's physician.

The last specimen presented was one of

ULCERATIVE ENDOCARDITIS,

with perforation of one of the aortic cusps. Long festoons of fibrin were found attached to the valves. An examination for bacteria showed the presence of the staphylococcus pyogenes aureus.

Dr. Mendelson, referring to the cause of sudden death in the case of aortic aneurism without rupture, said that he thought the explanation of death from compression on the trachea was not a satisfactory one. It was hard to suppose that a person would die so quickly, and without a struggle, from simple suffocation. He thought a more rational explanation was that death resulted from paralysis of the heart due to sudden inhibition from stimulation of the vagus nerve.

Dr. Prudden said that when death resulted from the presence of a foreign body in the larynx, the person often died very suddenly and without a struggle.

Dr. Biggs said that he had seen two or three such cases in children. In one case, death was caused by the lodgement of a collar-button between the two vocal cords, and in another by the presence of a screw in the larynx.

Dr. Mendelson said that this class of cases seemed to him but to prove the point he wished to make, for there were none of the usual signs of death by suffocation present. It was more probable that in them, too, death was caused by the reflex stimulation of the vagus through the laryngeal nerves, and consequent cardiac paralysis by inhibition. In the case of aneurism just described the irritation arose probably from the sudden distention of the sac, caused by straining at stool, stretching or rupturing some of the fibres of the recurrent laryngeal, or other branches of the vagus in intimate relation with the aorta.

Dr. Frank Ferguson presented a specimen of

VOLVULUS.

The patient had been seen by a number of physicians, but it was not thought advisable to operate.

Dr. H. J. Boldt said that the history did not make it clear why an operation was not performed, though he doubted not that sufficient contra-indications were evident to those who had the case in charge. It was a fact, however, that many cases of intestinal obstruction were allowed to go on to a fatal termination, which might have been averted by a timely operation. He had himself never seen a case of "idiopathic" peritonitis, and he believed that many patients, who would almost surely die if medicinal measures alone were relied upon, might be saved by early surgical interference.

Dr. John S. Thacher presented a unique specimen, consisting of

A SAC ENCLOSING THE SMALL INTESTINE,

which was discovered at the autopsy of a man dead of portal thrombosis. Some three years ago the patient had had a hemorrhage from the stomach, and another hemorrhage from the stomach and bowels had occurred about a month before death; otherwise the history was negative. The portal vein was found obstructed by a thrombus, but this had had nothing to do with the other condition. The sac enclosing the intestine was a thick, white, and glistening membrane. The only explanation that suggested itself to the speaker was that a knuckle of the duodenum had in some way been brought to make pressure on a portion of the mesentery; this had yielded a little, making a sort of pouch, and then more of the intestine had followed, the mesentery yielding still further, until in this way a complete sac had been formed.

Dr. Thacher then presented a specimen of

ABSCESS OF THE LIVER.

The patient had been ill for about five months with pains in the right hypochondriac region. At the time of his admission to the hospital he was coughing up some blood and pus, and continued to do so for some time. At the autopsy it was found that the abscess of the liver had ruptured through the diaphragm into the right pleural cavity. The right lung had been almost entirely destroyed by the pus.

Dr. Biggs asked whether the speaker had ever seen abscess of the liver occurring independently of ulceration in the cæcum or other parts of the intestine.

Dr. Thacher replied that he had seen a case of this kind very recently, in which no evidences of ulceration were encountered in any portion of the intestine.

Dr. Ferguson thought that intestinal ulcers were almost always present. Sometimes it would be found

that ulceration had existed, but that cicatrization had taken place, so that the evidences of the previous condition would be manifested only on careful search.

Dr. J. S. Thacher reported from the Committee on Microscopy concerning the specimens of inflamed and necrotic lung, and of intestinal ulcers from a child, presented at the meeting of October 24th, by Dr. Holt. Microscopical examination of sections of the lung showed the usual appearances of broncho-pneumonia, with an unusual massing of leucocytes in portions. No bacteria were found.

Microscopical examination of the ulcer of the intestine showed along all of the free border of sections an abundance of bacteria, rod-shaped, many slightly curved, some united so as to resemble spirilla. They appeared uniform, and at the site of the ulcer were found at a considerable depth in the inflamed tissue.

The Society then went into executive session.

Stated Meeting, November 28, 1888.

W. P. NORTHRUP, M.D., PRESIDENT, IN THE CHAIR.

DR. RICHARD VAN SANTVOORD presented some specimens showing

PRIMARY TUBERCULOSIS OF THE GENITO-URINARY TRACT.

The patient, J. G——, had been under observation at intervals for about three and a half years, in the Randall's Island Hospital, his main trouble being supposed to be chronic diffuse nephritis. The urine was generally of low specific gravity, frequently below normal in quantity, and usually containing a moderate amount of albumin. Casts of various sorts were found from time to time. Within two years the epididymis, first of one side, and, after a short interval, of the other, became swollen, hard, and finally suppurated. A sinus also formed over the sacrum, discharging pus. The strength, meanwhile, failed gradually. For about six weeks, during the last summer, he was troubled with frequent micturition and his urine was observed to contain considerable pus. Under treatment the symptoms subsided and the cystitis was supposed to have been cured. Attention was drawn to his lungs only a few weeks before death, which occurred November 10, 1888.

The autopsy disclosed a very emaciated body, with a sinus over the sacrum leading into the right sacro-iliac synchondrosis, whose bony surfaces were rough and carious. The cartilage of the left fifth rib was softened and necrotic, and communicated with an abscess under the pectoral muscles. The pia mater was thickened, and a single small tubercle was found on the convexity of the left side of the cerebrum. The heart was pale and slightly atrophied. The lungs showed firm gray, tubercular clusters throughout, some dilatation of the bronchi, rather recent pneumonic consolidation of left lower lobe, old adhesions over right lung, and recent ones over left.

Both kidneys were invaded by tubercles, affecting most extensively the pyramidal portions, but extending also in many places through the cortex. The tubercular material was for the most part cheesy, and in places was breaking down. The portions of the kidney not invaded by tubercle showed the cortex to be considerably atrophied, the markings indistinct, and the cut surface of a mottled yellow color. The right ureter showed two tubercular masses, one-twelfth of an inch in diameter where the ureter passed over the brim of the pelvis. In the left

ureter there were two small masses near the renal pelvis. The mucous membrane of the bladder was ulcerated away for about one inch around the urethral orifice. Back of this was a second ulcer, about three-fourths of an inch in diameter, and several smaller ones were scattered over the vesical surface. The prostatic urethra was extensively infiltrated, the process extending forward into the spongy portion of the urethra. Small tubercular masses also were scattered all along the urethra, in decreasing numbers as the meatus was approached. The prostate was extensively infiltrated and cheesy. The right vesicula seminalis formed a cheesy mass about $\frac{1}{2}$x$\frac{1}{4}$ inch in size. The left was slightly involved in the same process. The epididymis on each side was enlarged and cheesy. The body of one testicle was invaded by more recent gray tubercle. The other was normal. The intestines showed some enlargement of the solitary follicles and old adhesions. The liver was normal in size, but universally adherent, and on both the free and the cut surfaces a few small tubercles were observed. Diffuse cirrhosis, moderate in amount, existed.

The case seemed to have been one of primary tuberculosis of the genito-urinary tract, because the lesions, particularly in the kidneys and about the neck of the bladder, were much more advanced than those in the lungs. Their extraordinary latency was very remarkable. Prior to the development of symptoms pointing to cystitis nothing was discovered in the urine to suggest anything more than chronic nephritis, although it had been frequently examined. It did not seem possible to determine which began first, the nephritis or the tubercular kidney lesion. The former was positively known to have existed for three and a half years, and probably antedated the patient's first entrance into the hospital.

There was one question, Dr. Van Santvoord said, which he would be glad if any of those present could answer for him, and that was as to the rapidity with which such a process as this could be developed. In reply to a question as to the order in which the several organs became involved, he said that the most marked lesions were found in the bladder. The epididymitis had existed for about eighteen months, and it was probable that the process had begun in this part or in the prostate gland. The pulmonary lesions were evidently less advanced than those in the genito-urinary tract.[1]

[1] Since the above was written Dr. Van Santvoord adds the following: Ziegler (Path. Anat.) states that in the great majority of cases renal tuberculosis is embolic, but that in rare cases a tuberculosis beginning in the bladder, the prostate, the seminal canals, or the testicle may infect the kidney by way of the ureters (p. 1129, edition of 1885). On page 470 he calls attention to the identity of this mode of infec-

Dr. Walter Mendelson raised the question whether we were justified in unhesitatingly accepting this case as one of primary tuberculosis of the genito-urinary tract. It was true that the anatomical lesions seemed to show that the process had been longer at work in the genito-urinary tract than in the lungs; nevertheless, there was not, in his opinion, anything to show that the tubercular process might not have long been latent in the lungs, and that the genito-urinary tract had not been infected from them. This was rendered the more probable by the fact that we so often find on autopsy cheesy nodules in the lungs of persons who, during life, had given no signs of their existence. In view of the obscurity of the mode of infection in cases of genito-urinary tuberculosis, the speaker thought we should exercise the greatest caution in accepting the evidence on which the claim for primality was based in these cases.

The President said that, in his opinion, the first of the genito-urinary organs to become diseased in this case was the testicle or the epididymis. It was his experience that the kidney was usually the last of all the organs to be involved.

Dr. B. Farquhar Curtis referred to Finck's studies on the subject of genito-urinary tuberculosis, the results of which seemed to prove that the prostate was infected before the kidneys.

Dr. Henry P. Loomis presented a specimen showing

PECULIAR TUMORS IN THE HEART-MUSCLE.

The heart was from a woman, twenty years of age, a prostitute, who gave the following history: She had borne three children and had had two miscarriages, the last miscarriage being about a year ago. She had always been healthy, with the exception of an attack of intermittent fever one year ago. She denied ever having had syphilis. Four days before admission to the hospital she had had a chill, followed by fever and sweating, and this was repeated twice, the last chill being on the day before admission. Since the first chill she had been troubled with dyspnœa. She had had muscæ volitantes, diplopia, frontal headache, dizziness, and swelling of the feet and legs during her pregnancies, but none in the in-

tion with that due to parasites, which give rise to more acute forms of disease. The predominant affection of the pyramids in the case reported, as above stated, points to infection by way of the ureters rather than by embolism. It is of course quite possible that the pulmonary tuberculosis might have been due to independent infection, especially as the patient was for a long time an inmate of a hospital ward in which there were always several cases of pulmonary tuberculosis. Kaufmann (Deutsche Chirurgie) states that tuberculosis of the urethra is very rare, and is always accompanied by extensive tuberculosis of the genito-urinary organs. It extends from the bladder and affects usually the prostatic, rarely also the membranous, urethra.

tervals. Of late the urine had been increased in amount. On admission the physical signs were those of pneumonia. The heart-sounds were normal. The patient died on the third day after entering the hospital.

On autopsy, the entire right lung was found in the third stage of pneumonia. There were also evidences of fresh pleurisy. The liver was normal. The spleen was greatly enlarged. The kidneys presented the lesions of chronic diffuse nephritis. Pyosalpinx and purulent oöphoritis were present, and on the cervix uteri was what seemed to be a chancroid. No syphilitic lesions were found, however, in any other part of the body.

The heart was normal in size, no change was evident in the cavities or the valves, a few small atheromatous patches were noticed in the aorta, just above the valves. Two tumors the size of a French olive were embedded in the wall of the left ventricle near the auricular-ventricular septum, one an inch below the aortic valve, the other below and about an inch from the apex. The tumors were circumscribed, of a whitish-yellow color and hard consistency. A section of one of the tumors showed under the microscope that the centre was in a condition of coagulation necrosis, and that the peripheral zone was made up of round cells, between which was seen a basement substance. The round cells extended for a short distance beyond the apparent edge of the tumor in between the muscle fibres.

Dr. Prudden, who examined the section carefully, expressed the opinion that the tumor was a *gumma*. Of the tumors of the heart tubercle is the most common, while gummata are very rare. The speaker had found but a few cases mentioned in American or English literature, and these were almost always associated with gummata in other organs.

Dr. Mendelson suggested that the lesion might possibly be the result of infection from the pneumonia, and he thought it would be well to examine the specimen for pneumococci.

Dr. Loomis replied that the lesion evidently antedated the pneumonia. The process was not an active one, and there was no acute suppuration present.

Dr. Van Santvoord said that it was not uncommon to meet with cases of endocarditis caused by the pneumococcus, but in the specimen presented the process was apparently much older than the pneumonia. The gross appearance of the lesions certainly suggested a gumma.

Dr. T. H. Myers presented some specimens of

UNIVERSAL SUPPURATIVE ARTHRITIS.

Cases, he said, where two, or even several joints are involved in such a process are common enough, but in

a search through the literature of the past ten years, he had found no case of a similar *universal* joint disease.

The specimen was taken from a foundling two months old. The autopsy was made by Dr. William P. Northrup. Body well nourished; slight swelling over carpo-metacarpal joints of left hand. Brain normal, as were lungs and spleen. Liver dark, congested, slight mottlings (anæmic?), in size normal. Gall-bladder moderately distended. Kidney cortex pale, markings indistinct. Stomach empty, mucous membrane pale, amount of mucus increased. Intestines empty, mucus increased in amount, bile-stained, in stringy masses; mucous membrane pale. Bladder normal. Joints: Right sterno-costal, both temporo-maxillary, both scapulo-humeral, both elbow, both wrist, both carpo-metacarpal, both hip, both knee, both ankle, both tarso-metatarsal joints contained abundance of pus, which was also found along the tendons of all the fingers and toes. The cartilages were easily broken from the bones. The cancellous tissue of the latter was deep red, soft, and could be easily broken down with the thumb.

Unfortunately, in this case the history of the parents and the details of parturition could not be obtained, as the child was a foundling. From the fact that syphilis was suspected before death the speaker felt sure that had any of the visceral lesions of syphilis been present they would have been noted prominently, also that no suppurative condition of the umbilicus or umbilical vessels would have escaped observation. The child, when admitted to the asylum, had onychia. The left hand was reddened and swollen, as was also one foot. The baby was sore all over on handling and was thought to have syphilis. It was removed to the "Marasmus Ward," and died four days later, without presenting any other symptoms. On disarticulating the sternum and clavicle, thick pus was noticed escaping from the joint and this caused the methodical examination of the remaining joints, as detailed above, to be made.

In looking over the literature of the subject the speaker had found the case most nearly approaching this in the number of joints involved to be one reported by Dr. Huber (*Arch. f. Path. Anat.*, Berlin, 1882, lxxxviii, p. 246). At the careful autopsy made by him on this case—a premature child fourteen days old—nearly all of both the large and small joints contained laudable pus; the ligaments, bones, and cartilages were normal, the synovial membranes infected in parts only. There was œdema of the lungs; the navel and umbilical vessels were normal. There were only a few swollen glands at the lower part of the ileum. The pleura, pericardium and peritoneum were

normal. He mentions, however, a bed-sore, a subcutaneous abscess on one foot, a mediastinal abscess, and a hæmatoma of the dura, with adhesions of dura and pia and a congestion and softening of the cerebral substance. The kidney contained a deposit of urates. The heart and other organs were normal. The mother had had an attack of acute articular rheumatism during confinement, followed by a relapse. As there was no history or appearance of syphilis, though a careful examination was made for this on both parents and child, nor yet of any of the exanthemata, Huber made the diagnosis of rheumatic arthritis. Dr. Myers thought this case was more like a pyæmia, either "spontaneous," or secondary to the hæmatoma or bed-sore.

Another case is reported by Lane ("Tr. Path. Soc. London," 1884–5, xxxvi., 343). Here there was purulent effusion into several joints in a child certainly syphilitic. The joints affected were the hip, the shoulder, the right knee, and both elbows. They contained an abundance of pus. The synovial membranes were very vascular. The remaining joints were normal. Lane noted no change in the ossifying layer of the cartilage, nor in the articular cartilage. The child was healthy till six weeks old, when an eruption and snuffles were noted, and emaciation became marked, the child dying twenty days later. The writer considered the acute synovitis as corresponding with the prodromal form of synovitis found in adults, which precedes the first eruption, disappearing when this has become general.

Dr. Lee (*Brit. Med. Jour.*, November 6, 1880, p. 743) reports a similar case, cured by antisyphilitic treatment.

In considering the diagnosis in this present case, Dr. Myers said that it lay between syphilis, rheumatism, and pyæmia. In favor of the syphilitic origin was the præmortem diagnosis, and the fact that such a case is possible. On the other hand these congenital syphilitic joint-lesions are usually confined to a *very few* of the *larger* joints and are at best very rare. The epiphyseal line, or the periosteum, are the usual seats of the disease. These joints where the epiphyseal line runs within the joint limits, as the knee and elbow, are involved by preference.

Against the rheumatic origin was the extreme rarity of such cases in very young infants. Eustace Smith says ("Dis. of Children," N. Y., 1884, p. 155) the younger the child the more likely will the disease fasten upon the heart to the exclusion of the joints.

Taylor ("Dis. of Osseous System in Children," N. Y., 1875, p. 150), denies the existence of rheumatoid arthritis in infants, though recognizing it in children.

Howard ("Pepper's Sys. of Med.") regards acute articu-

lar affections in sucklings syphilitic or pyæmic as a rule, but recognizes a few authentic cases of rheumatic origin.

Widenhofer met with but one case (an infant twenty-three days old) out of a service of 70,000, and Rauchfuss in 15,000 cases found but two occurring in children at the breast (Senator, "Ziemssen's Cycl. of Pract. Med.," xvi., 17). There is usually, also, in these cases redness, swelling, and pain. The inflammation jumps from one joint to another after four or five days, and, finally, suppuration is very rare.

J. Lewis Smith, however ("Dis. of Children," N. Y., 1886, p. 399), describes briefly a form of rheumatic arthritis in young children which is not marked by redness, swelling, or much pain. The number of joints involved, however, is small. Cornil and Ranvier also ("Path. Histology," Phil., 1880, p. 228) describe cases of acute rheumatism, where the inflammation has attacked several articulations or only a single one, and the articular cavity is filled with a creamy pus, analogous to that of an abscess.

Pyæmia is the most usual cause for multiple suppurative arthritis, but though several joints are often involved the speaker did not know of another case where all were. Of this condition Billroth ("Surg. Path.," N. Y., 1883, p. 344) writes: "Purulent articular inflammation occurs in the pyæmia of the newly born. Children are even occasionally born with them, as has been witnessed by myself and others. Inflammations may develop and even run their course during fœtal life, as is shown by the cases where children are born with joints fully developed, but anchylosed."

The case seemed to belong among what Delafield and Prudden ("Handbook Path. Anat.," N. Y., 1885, p. 513) call "spontaneous pyæmia," where, without wound or fracture and without visceral infarctions, inflammation of joints and serous membranes occur with all the symptoms of pyæmia. Such cases must be very rare, as in the lungs alone metastatic abscesses are found in ninety-one per cent. of pyæmic deaths, according to Bryant's tables ("Surgery," Phil., 1881, p. 58).

A gonorrhœal origin had been suggested, the infection perhaps occurring from an ophthalmia. But there was no record of such an ophthalmia in this case, though the eyes of these children are always examined. The number of joints involved would also militate against this idea, though Fournier states that temporo-maxillary, sacro-iliac, sterno-clavicular, and intervertebral joints are affected in this disease as well as the larger joints ("Nouvel Dict. de Med. et de Chir. Prat.," t. v., p. 230).

The purulent arthritis described by Thomas Smith ("St. Bartholomew's Hospital Reports," 1874, p. 189) as origi-

nating from "subarticular abscesses," and occurring in such a large percentage of cases in infants under two months old, was negatived here by the absence of such abscess-cavities and because this disease only involved one joint or two.

The general involvement of joints and negative history would also exclude the form of purulent arthritis in children, called by Volkman "catarrhal inflammation."

The diagnosis seemed, then, to be, by exclusion, spontaneous pyæmia.

Examination of the specimens showed smoothness of the articular surfaces; no anchylosis or periarticular thickening; no subarticular abscess-cavities; no change apparent to the eye in epiphyseal cartilage; no thickening of periosteum; no metastatic abscesses. The valves of the heart were smooth.

The President remarked that cases such as this must be very rare, he himself having never seen or heard of an instance in which so many joints were involved. There were no signs of syphilis about the child, nor was there any suppuration in the umbilical vein.

In reply to the question as to whether the pus in the joints had been examined for bacteria, the President said that no examination of the kind had been made, as the case had been seen several years ago, at a time when the search for bacteria was not as eager as it was at present.

Dr. John S. Ely presented a specimen of

ANEURISM OF THE ARCH OF THE AORTA: SUDDEN DEATH WITHOUT RUPTURE OF THE SAC—REMARKS ON THE CAUSE OF SUDDEN DEATH.

The patient was a woman, fifty-five years of age, a native of Ireland, and a washerwoman; admitted to Bellevue Hospital toward the end of October. For five or six years she had noticed occasional swelling of the feet. For six months she had suffered from dyspnœa on exertion, which steadily became worse. Cough for about a month. Two weeks prior to admission she had noticed a tumor at the root of her neck. Dysphagia had been troublesome for six weeks or so, and there had been some pain in the region of the tumor.

Physical examination disclosed slight emphysema and bronchitis; somewhat rapid and feeble heart, but no evidence of valvular lesion; liver and spleen normal in size. At the root of the neck on the right side, just above the sterno-clavicular articulation, there was a soft, compressible, pulsating tumor about the size of a small hen's egg. The pulsation was expansile in character, but there was

neither thrill nor bruit. Pulse in both radial arteries was equal; also in both carotids. There was very marked atheroma of the arteries. Urine showed no albumen, but a few casts and pus-cells. Laryngoscopic examination disclosed paralysis of the right vocal cord.

Patient remained in much the same condition, with considerable dyspnœa and some cough, until the night of November 12th, when she fell out of bed in her sleep. She was helped back to bed by the nurse, who left her apparently none the worse for her mishap. In about fifteen minutes the nurse returned and found the woman dead, lying flat on her back, with no indications that death had been preceded by either pain or convulsion. The inference was that the aneurism had burst.

At the *autopsy*, performed fifty-four hours after death, the body was found to be well nourished. The brain was not examined. On opening the thoracic cavity a tumor about $3\frac{1}{2}$ inches by $2\frac{1}{2}$ inches was discovered, partly adherent to the manubrium of the sternum, and consisting of a dense sac of fibrous tissue containing an organized clot of considerable size, and some serous and bloody fluid. This tumor was found to originate from the arch of the aorta, where the ascending portion passes into the transverse. The innominate artery was incorporated in the posterior wall of the sac. The pneumogastric nerve of the right side was much flattened and involved in the wall of the sac. The phrenic and recurrent laryngeal nerves were also pressed upon by the tumor. The ascending portion of the arch of the aorta was very much increased in size.

Heart was somewhat hypertrophied, flabby, and showed no lesion on the valves. Lungs were normal, with the exception of much pigment. Liver was normal in size, but showed slight congestion and fatty degeneration. Spleen was hard and small. Kidneys were both rather small; capsules adherent; cortex thin, markings indistinct. Whole kidney quite red.

There was very marked endarteritis in all the main arteries, and an organized clot was found in the left internal jugular vein about the middle of the neck. There was no rupture of any vessel nor of the aneurismal sac.

The problems connected with the cause of sudden death are discussed with such vagueness in the text-books that I have thought it might not be uninteresting to the Society if I were to use this case as a text for presenting to its consideration some of the physiological factors involved, with a view to directing attention more rationally to the investigation of such cases.

It was long ago discovered that the heart of a frog could be removed from the body and still continue to beat rhythmically for a very long time, if kept in a warm,

moist chamber. Latterly, Martin, at Johns Hopkins University, has shown that the heart of the dog may be completely cut off from all connection with the cord and brain—isolated, as they call it in Baltimore—and still continue to beat regularly for several hours, provided only that its nutrition be kept up by a continuous supply of defibrinated dog's or calf's blood.

These experiments show that the essential elements of the heart-beat are situated in the heart itself, in the frog and dog at any rate; and thus far, there is nothing known in human physiology which renders a similar arrangement in man improbable.

Investigation has shown that the parts of the heart necessary to its activity are the intrinsic ganglia and the heart-muscle. The heart of a frog may be cut up in small pieces, and the portions containing ganglia will continue to contract rhythmically, while the portions destitute of ganglia are perfectly quiescent, thus proving that the ganglia are essential to the beat. We must then look to the heart itself for the source of the trouble in cases of sudden death due to paralysis of the heart.

But may not sudden death be due to some other process than paralysis of the heart? I think not. I cannot conceive of it as the result of any other process than sudden stoppage of the heart, and I cannot conceive of sudden stoppage of the heart as the result of anything else than paralysis of the heart. It is surprising that "spasm" of the heart should still be spoken of in the text books. Physiologists have never been able to produce spasm of the heart, and pathologists have not shown how any such phenomenon is possible. Still, Gairdner, in the article on "Angina Pectoris" in "Reynolds's System of Medicine" (vol. iv. p. 579, 1877), says that opinion as to the cause of death in that disease is divided between "spasm" and paralysis of the cardiac muscle. In direct antagonism to such a theory of "spasm" of the heart, physiology teaches that each contraction of the cardiac muscle is simple, like the contraction of ordinary striated muscle caused by a single galvanic shock. The interrupted current when applied to heart-muscle produces, not a continuous ("tetanic") contraction, but a series of single rhythmic contractions. In other words, the heart cannot be put into a state of "spasm" by means of the most powerful neuro-muscular stimulant known to us, electricity. Let us then rule out "spasm" as a possible explanation of sudden stoppage of the heart, and consider the causes and effects of paralysis of that muscle.

Marked differences in the physiology of heart muscle and ordinary muscle certainly exist, but still there seem to me to be sufficient points of similarity to make the

pathology of the one very useful in the elucidation of the pathology of the other.

The essentials to voluntary motion have been found to be (1) the nerve-cells in the cortex of the brain, (2) the paths of communication between these and the muscles, and (3) the muscles themselves; and decided lesion in any one of these is attended with paralysis. In the case of the heart, we have seen that the intrinsic ganglia and the muscle are essential to the heart-beat, and we must of necessity suppose paths of communication between these elements. Reasoning, then, by analogy, we must conclude that paralysis of the heart would result from lesion in the ganglia of the heart, in the paths of communication between these and the heart-muscle, and in the muscle itself.

There are, then, theoretical grounds for the belief that lesions in the intrinsic ganglia and in the heart-muscle may be sufficient cause of paralysis of the heart. What has pathological investigation advanced in substantiation of this theory? Owing to the difficulty in finding the intrinsic cardiac ganglia but few observations of their disease are on record. Iwanowsky [1] has published the results of his investigation of the changes which occurred in them in a case of typhus fever. The nerve-cells in the ganglia were swollen and opaque, with indistinct nuclei; and in some spots well-marked degeneration of the cells was observed, as well as slight changes of an inflammatory nature in the connective-tissue stroma of the ganglia. Wassilieff [2] found decided changes in the same structures in hydrophobia. But the most interesting cases have been reported by Putjatin.[3] Only five cases of heart disease were investigated, but in all of them marked lesions in the intrinsic ganglia were found. The first of his cases bears directly upon the point under discussion. It was that of a man about forty years of age, in whom no lesion could be found to account for his sudden death except those in the intrinsic cardiac ganglia. The heart-muscle in this case is said to have been normal. The lesions found by Putjatin in these cases were hyperæmia, increased production of connective tissue, fatty and pigmentary degeneration of the ganglion cells, and in one case calcareous deposit in the connective-tissue stroma.

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[1] Iwanowsky: Zur pathologischen Anatomie des Flecktyphus. Journal der normalen und pathologischen Histologie und der klinischen Medicin. 1876.

[2] Wassilieff: Ueber die Veraenderungen des Gehirns und der Herzganglien bei der Lyssa. Centralblatt f. d. medicin. Wissenschaften, 1876, No. 36.

[3] Putjatin: Ueber die pathologischen Veraenderungen der automatischen Nervenganglien bei chronischen Herzkrankheiten. Virchow's Archiv, lxxiv. (1878), 461.

Although these are but few observations, too few alone to be sufficient foundation for a theory, still, in substantiation of a plausible theory deduced from other data, they are of the greatest importance.

Paralysis of the heart as the result of inflammatory and degenerative changes in the cardiac muscle, as, for example, the interstitial myocarditis so commonly found in cases of angina pectoris, is matter of too common observation to warrant further notice here.

But, it may be asked, if sudden death is simply due to paralysis of the heart-muscle, how is it that this comes on suddenly, paralyses in the skeletal muscles being so often slow in development? I answer that I do not consider paralysis of the heart to be always developed suddenly—far from it—and that in paralyses of the skeletal muscles we have occasionally a very rapid onset, for example, in infantile paralysis. Further, the conditions in the two cases are dissimilar. Whereas in the skeletal muscles the resistance to be overcome varies within very wide limits, in the heart-muscle it is practically always the same, variations in arterial pressure being comparatively very slight. In the skeletal muscles the power to move the limb is usually preserved long after distinct weakness can be determined, but usually there comes a time when this power to move the limb is lost too, and that suddenly. In the heart-muscle the rhythmic beat continues until a moment comes when the resistance can no longer be overcome, and when the heart-beat must of necessity stop suddenly.

It remains now to mention certain extrinsic processes to which sudden stoppage of the heart has been attributed.

There are in the central nervous system two mechanisms regulating the heart-beat, the one accelerating, the other inhibiting. Of the accelerating mechanism it is not necessary for us to say more than that its sole function, so far as known, is the increase in frequency of the cardiac contractions, and that it consequently has nothing to do with the subject under discussion. The inhibiting mechanism is, on the other hand, closely connected with it. The centre of this mechanism is located in the medulla oblongata, and is connected with the heart through the vagus nerve. Stimulation of the centre or of the vagus causes slowing of the heart-action, but, in order that complete stoppage be produced, the stimulus applied to the vagus nerve must be of very considerable strength and must be suddenly applied—very gradually applied stimuli, though of considerable strength, being ineffectual. For this reason it is thought that sudden stoppage of the heart as the result of stimulation of the vagus nerve by

tumors must be extremely rare. In these cases the pressure upon the nerve must increase so gradually that entire disintegration of the nerve may be produced without sufficient stimulation to cause inhibition of the heart. In connection with the consideration of stoppage of the heart as the result of stimulation of the inhibiting mechanism, it must be borne in mind that the stimulus may be applied to the centre reflexly. The sudden stoppage of the heart obtained by striking a frog upon the belly is a familiar example of this, and all sudden death as the result of "shock" must be explained in this way.

The recurrent laryngeal nerve is an important path along which impulses may pass to the inhibiting centre in the medulla oblongata, producing sudden inhibition of the heart reflexly. This is, I think, the explanation of those cases of sudden death following instantly the lodgement of a foreign body in the larynx, to which reference was made at the last meeting.

In conclusion, then, I am of the opinion that the lesions which may cause sudden death should be looked for in, 1, the cardiac muscle; 2, the intrinsic cardiac ganglia; 3, the inhibiting centre in the medulla oblongata; 4, the vagus nerve; and, failing to find any adequate lesion in these parts, the possibility that death may have been caused by reflex stimulation of the inhibiting centre is not to be lost sight of.

Dr. John Messenger recalled a case of an enormous aneurism of the aorta, the specimen of which he had presented to the Society some twenty years ago. There was no rupture of the aneurism, but the man died in great distress. He was two months in dying, suffering all that time from extreme dyspnœa and cough. At the autopsy adhesions were found between the aneurismal sac and the sternum. The lungs were markedly compressed by the tumor.

Dr. H. P. Loomis said that he believed that some of these cases of sudden death from aneurism could be explained by an examination of the heart-muscle. In a case of this kind which he had seen some time ago the heart seemed to be normal, but under the microscope the muscle was seen to be extensively diseased.

Dr. William P. Northrup presented a specimen, showing

EXTENSIVE PULMONARY INTERLOBULAR EMPHYSEMA,

with the following history:

The patient, a female child of five months, was brought to the Out-door Department of the New York Foundling Asylum, by the nurse, who spoke only Italian. It is the habit of the Italians to return a child immediately if it

becomes sick. She said the child was "little sick," and she was certain it had not whooping-cough and had never had convulsions.

The child coughed moderately, had no general depression, was well nourished, played about the room and laughed. The diagnosis of ordinary bronchitis of mild type was made, and nurse and patient sent home.

Fourteen days later patient was returned to the hospital with a temperature of 103°, with severe dyspnœa and cyanosis. It died three days later, or on the seventeenth day after the first observation was made.

Autopsy.—Body was well nourished, fat; brain normal; larynx moderately congested and dusky; mucous membrane wrinkled; aryteno-epiglottidean folds swollen and wrinkled; vocal cords normal; no ulcers in larynx; mucous membrane of epiglottis and trachea moderately congested and dusky. The mucous membrane had the appearance of being wrinkled, from post-mortem removal of a portion of the œdema and congestion.

Lungs: The left was the seat of extensive interstitial emphysema of the upper lobe. Beaded rows of air-sacs radiated from the root to the anterior margin, separating the lip of the lung so as to allow light to shine through from one lateral surface to another. One vesicle was larger than its fellows and separated the anterior upper lip of lung more than a centimetre, and bulged forward like a bladder of air under moderate pressure. At the posterior margin of the lower lobe was another air-bladder of simi lar size. In the posterior portion of the apex was recent pneumonia, extending about half way from apex to root, having upon the surface numerous petechiæ.

Right lung, moderate pneumonia of posterior margin from apex to base; petechiæ on pleural surface and few scattered vesicles of emphysema.

Stomach and intestines were the seat of moderate catarrh. Other organs normal.

This case had no previous history to explain the cause of the lesion. The condition of the larynx indicated that there may have been œdema and spasm, but there was no history of it, and the case was under the observation, for the last three days of life, of Dr. Lynde, the house physician, and no whooping-cough nor spasm of the glottis was noted.

In this connection it was of interest, Dr. Northrup said, to refer to a similar case published in the *American Journal of the Medical Sciences*, July, 1883, entitled "Extensive Interlobular Emphysema and Abscess of the Lung, after Whooping-cough, in a Child of Two Months—Unique Case." The case was published by the speaker and was considered, at that time, "unique." He had

since seen a specimen very like it in the Museum of the Royal College of Physicians, in London. These were the only cases of such extensive emphysema of which he was aware.

Dr. Northrup also presented a gross specimen, together with microscopic slides, demonstrating a case of

NECROSIS OF THE LUNG

(posterior marginal), in a case of pleuro-pneumonia and cerebro-spinal meningitis.

Roseman B——, aged two and a half months, was reported by the wet nurse to be "sick," but without any special symptoms. She nursed well, had no vomiting or diarrhœa, was somewhat restless, had slight cough and a temperature of 102° F. Two days later, temperature 104° F., respirations rapid, dulness over right chest posteriorly, abundant fine râles. Five days later, or seventh day of illness, the right side of chest was flat from apex to base over posterior half of lung, and bronchial breathing was present from root to base. Liver was depressed five centimetres below the free border of the ribs in the nipple line. Temperature 102° F. Respiration 60, painful, general pain on handling. Ninth day child refused to nurse, became fretful, with 103° F. temperature, pulse 160, and respirations 60. Anterior fontanelle prominent, pupils normal, no paralysis, no convulsions, head was drawn somewhat backward but not rigid. Failed rapidly and died on the tenth day. (Records of the case furnished by Dr. Geo. S. Lynde.)

Autopsy, fourteen hours after death.—Body poorly nourished; brain and cord covered with extensive pus and fibrin exudate, veins distended with blood and having upon their lateral margins borders of pus and fibrin. The exudate upon the cord was mostly upon the posterior surface. Ventricles were softened post mortem, velum interpositum covered with a distinct layer of pus and fibrin. Lungs: Right pleural cavity contained a jelly-like pad of pus and fibrin. This was an inch thick in the postero-lateral region, and wholly enveloped the lung except on its mediastinal aspect. The lung was compressed, and had in its lower and middle lobes a moderate amount of broncho-pneumonia. Along the posterior margin was a mass of lung tissue of lighter color, firmer, more smooth upon its cut surface than the surrounding lung, four centimetres in length, two in width, and one centimetre in depth. It had the appearance of necrosis from infarction. Besides the marginal mass there were several scattered masses having much the same appearance. In and about the necrotic areas were small abscesses or dilated bronchi, containing pus. These proved on microscopic examina-

tion to be collections of pus and fibrin in the interlobular connective tissue. Heart, endocardium, pericardium, peritoneum, liver, spleen, stomach, and intestines normal. Kidneys not congested, no areas of congestion or anæmia—appeared normal.

Microscopic examination.—The marginal area consisted of necrotic tissue. Skirting this on its pulmonary side was an irregular zone of tissue congested, having beside tortuous vessels, excessive exudation of fibrin and pus with abundance of blood-cells. The connective-tissue interlobular septa were densely packed with pus and fibrin, forming what appeared to be small abscesses or dilated bronchi referred to in the description of the gross appearance. No thrombosis was found. There was evidence of severe exudative inflammation in and about the necrotic area. At the posterior pleural margin there was found necrosis of the lung-tissue. The necrotic tissue, stained by Gram's method, showed micrococci distributed in the tissue and within the pus-cells.

Dr. Van Santvoord thought the case looked like one of pneumonia from inhalation of a foreign body (Schluck-pneumonie).

Dr. Northrup said that he had found no such cause present in this case.

Dr. H. M. Biggs said that we do at times have such appearances as these in cases of foreign-body pneumonia. He had seen them occasionally at autopsies of idiots. He believed that, in these cases, micro-organisms were taken in with the foreign bodies, particles of food, or may be of fluid.

Dr. Northrup remarked that, in the case presented, one lung was normal, and would not this argue against the supposition of foreign-body pneumonia?

Dr. Biggs replied that it certainly must be very rare to have a foreign-body pneumonia confined to one lung. Referring to the specimen of interlobular emphysema, he said that he had never seen such a marked case in the human subject. He had recently, however, seen a very pronounced case of the kind in a cow.

The Society then went into executive session.

Stated Meeting, December 12, 1888.

W. P. NORTHRUP, M.D., PRESIDENT, IN THE CHAIR.

DR. JOHN S. THACHER presented a heart showing

TRICUSPID STENOSIS.

It was removed from the body of a woman, twenty-nine years of age, who died in St. Luke's Hospital. She had been married six years, and had had two miscarriages but no living children. There was no history of rheumatism, but the patient had had scarlet fever in childhood. She had had leucorrhœa for a number of years. There was no history of syphilis. Five months ago she had some swelling of the feet, and of the left leg and thigh, and three weeks ago the abdomen began to enlarge, and the patient suffered from dyspnœa. The face was congested, but there was no pain about the heart, no palpitation, and no headache. The urine had a specific gravity of 1.010, and contained a trace of albumin, some hyaline and granular casts, and leucocytes. Examination of the heart showed the apex-beat in the fifth intercostal space, four and one-half inches to the left of the median line. The heart's action was irregular. A loud murmur was heard at the apex, but loudest over the mitral area; it was thought to be presystolic, and was not transmitted. There was, also, a loud systolic murmur at the base, loudest over the aortic area, and transmitted upward. A few sibilant and sonorous râles were present. The abdominal cavity contained fluid.

Autopsy.—The pericardial, pleural, and peritoneal cavities contained fluid. The left lobe of the liver was twice the size of the right, and the latter showed some cicatrices, was slightly hobnailed, and seemed to be cirrhotic. The kidneys showed many scars, probably from infarcts; the cortex was thickened, somewhat pale, and the markings were a little indistinct. There was stenosis of the mitral, aortic, and tricuspid valves. The tricuspid would not admit two fingers, was smooth, circular, and slightly funnel-shaped. The mitral admitted one finger, and was very similar in appearance to the tricuspid. The aortic valve would not admit one finger, was nearly circular in shape, and nodular.

Dr. Thacher also presented another specimen, showing a

CONSTRICTION OF THE INTESTINE,

produced by an inflammatory adhesion connecting two portions of the mesentery. The condition was discovered

only at the autopsy, as the constriction was not sufficiently tight to give rise to any symptoms during life.

Dr. John C. Peters referred to the frequency of the occurrence of valvular disease of the heart after scarlet fever, which was much greater than was commonly supposed. He had seen many such cases.

Dr. Herman J. Boldt presented a monstrosity consisting of

A DOUBLE-FACED HEAD, WITHOUT TRUNK OR EXTREMITIES.

The specimen was a mass which was expelled from the uterus of a woman, thirty-three years of age, immediately after the birth of her sixth child, and prior to the expulsion of the placenta. In its descent it appeared to the touch as another head, and it was believed by the midwife in attendance that a second child was about to be born. After the delivery of the mass and the placenta, which was unusually large, an examination showed that the child and the monster had membranes and placenta in common, but each had its umbilical cord. The cord from the child had a central attachment to the placenta, while that from the monster had a marginal attachment. The child was a normal one.

The specimen measured three and three-quarter inches in the long diameter, two and three-quarter inches in the transverse diameter, and three inches in the vertical diameter. The weight was not taken. On close inspection the monstrosity had the appearance of a head with two imperfectly developed faces. The faces were on the inferior aspect of the mass, their crowns pointing in opposite directions. In one face two sulci were seen, and were thought to represent the seat of the eyes; also a small, pedunculated, fleshy growth, possibly a rudimentary nose. There was neither mouth nor ears. The other face showed one deep slit, oblique in direction, and a shallow sulcus as representatives of the eyes. The nose was a pedunculated piece of flesh, the size of a hazel-nut. The mouth was well marked, with an upper hare-lip and a well-developed lower lip. Starting just above the eyes was a growth of very fine hair, which was not present on the other face.

Examination by transmitted light showed the outlines of cartilaginous plates, which, it may be assumed, were destined to be two frontal and two parietal bones. These plates had dark spots, suggesting centres of ossification. The space between the upper margins of the plates included a greater part of the superior surface of the mass, and. especially in two places, the skin was very thin and transparent. The umbilical cord was on a plane midway between the faces, but a little to one side of a line drawn

from nose to nose. In looking over the literature of monstrosities Dr. Boldt had been able to find no record of a similar case. It seemed evident that this one was a double-faced head without a trunk.

Dr. William A. Hume, presented some specimens illustrating a case of

COMPOUND DEPRESSED FRACTURE AND LOSS OF SUBSTANCE OF THE FRONTAL BONE—ENCEPHALITIS—LATE EPILEPSY—DEATH.

A man, aged thirty, was injured, May 10, 1887, by the explosion of a steam-drum over which he was working. The heavy pieces of metal struck his forehead, lacerated the skin, and fractured the frontal bone just above the orbits. The fractured bone was comminuted and depressed, the membranes of the brain were lacerated, as was also a portion of the cortical substance of the right frontal lobe, and the superior longitudinal sinus was opened, causing considerable hemorrhage. There was little or no shock, and the patient did not lose consciousness. The depressed fragments of bone were removed, and the projecting sharp edges of bone remaining intact were cut off with rongeur forceps. The loss of bone substance was considerable, the opening being nearly oval in shape and measuring, at widest points, seven centimetres laterally and three centimetres from below upward, it was situated somewhat obliquely, on the right a little above right orbit, from which point the lower border extended downward and to the left, crossing nasal eminence and involving the left orbital plate. The loss of bone substance was about equal on either side of the median line, so that both frontal lobes of the brain were exposed.

When all depressed bone was removed, the hemorrhage from the superior longitudinal sinus ceased spontaneously, probably from the formation of a thrombus within the sinus. The edges of skin wound were sutured together and union by first intention took place; but later, from pressure of brain, several ulcerations occurred along the line of union, the entire line gave way, and brain substance protruded through the wound, completely filling the opening in the bone, and projecting externally to the extent of from one to two centimetres. Over this hernia cerebri compresses and bandages were applied, and so, by gradual pressure, the brain substance was replaced. The skin wound healed by granulation. Notwithstanding the injury to the brain and to the membranes, the head symptoms during treatment were slight; the patient was delirious once, but only for a short time, and the temperature was not much above normal.

For several months the patient seemed to have made a

complete recovery, and during that period the late Dr. Sands exhibited the case to the New York Surgical Society; but eight months after his injury the patient had an epileptic attack. The paroxysm was not severe, he was given bromide of potassium, ten grains three times a day; this quantity was gradually increased to twenty grains four times daily, but this amount producing "bromism" it was reduced to ten grains four times a day, and so continued. The epileptic attacks recurred at intervals of about eighteen months, not more than one attack at a time, and, as far as could be learned, were never limited to one side of the body; it is probable, however, that a local spasm first occurred, which very quickly became general, and all parts of the body were convulsed. The sense of smell was not affected, as might be supposed from location of brain lesion (to be described later), the olfactory nerve with its bulb being on either side of the longitudinal fissure, upon the under surface of each anterior lobe. The patient very seldom had headache, and never experienced an epileptic aura.

Ordinarily the skin covering the opening in the bone would advance and recede with each pulsation of the brain, but this was not so during a fit. I had an opportunity to observe the head-wound in one of his paroxysms. The skin was firmly drawn back through the bone opening, and the pulsations were no longer visible. This condition remained unchanged until the period of stupor, when the pulsations gradually became more and more pronounced, and, as consciousness returned. the skin projected a little beyond the level of the surrounding integument; its usual position was slightly sunken. It seems to me this fact is of value in support of the supposition that during an epileptic attack all but the initial phenomena result from vaso-motor disturbance, the cerebral arteries are contracted, and consequent anæmia of the brain ensues. We seldom have any indication, through so large an opening in the head, as to the changes in the intracranial blood-vessels during an epileptic attack. Küssmaul and Tenner have made observations to prove that during an attack of epilepsy the brain is anæmic, and it is a well-known fact that, both in animals and in man, loss of blood may be followed by convulsions. Three months before death the epileptic seizures increased in frequency and in violence, occurring about once a week with, often, two or three attacks at a time. The post-mortem examination of brain gave evidence of recent lesions to account for this. The intellect was not at all impaired until this time, when he had marked failure of memory. This was probably due to the frequency of the attacks. During the last week of life the fits were of

nightly occurrence, and during the twenty-four hours preceding death the patient had sixty paroxysms. They occurred in such rapid succession that before he could recover consciousness from one fit another had commenced, and in this condition, "status epilepticus," he died, November 10, 1888. At autopsy the margins of bone were rounded off by absorption. Firm adhesions existed between the skin, membranes, and brain. The dura had not entirely reformed, and the anterior portion of the right frontal lobe was uncovered by it. The pia over the anterior portion of the right frontal lobe was entirely obliterated, and the anterior part of each frontal lobe was softened and disorganized, but particularly marked in right lobe.

I wish to express my indebtedness to Drs. E. C. Spitzka and N. E. Brill for their kindness in making post-mortem examination of the brain. Dr. Spitzka furnished the following notes as the result of such examination, and cross-section diagram showing the lesion in the right frontal lobe. The diagram shows the posterior face of a section anterior to the anterior end of the caudate nucleus.

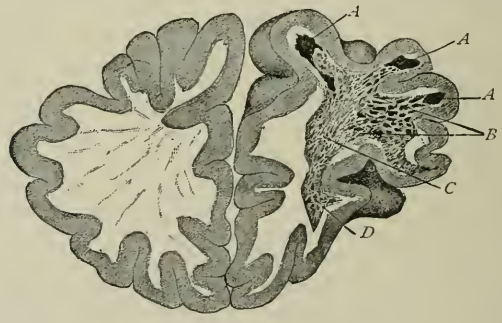

FIG. 3.—*A*, Cavity; *B*, large meshed area; *C*, fine meshed area; *D*, apparent recent lunar softening.

"Over the anterior part of the convexity of the right frontal lobe, in a line running in the frontal plane, convex upward for four and a half centimetres, then dipping down acutely for two centimetres, the dura and pia, indistinguishably fused, appeared drawn into the cerebral substance. The line in question corresponds nearly to the course of the gyrus frontalis marginalis. At and below it the fused membranous investment was firmly united to a trabecular tissue, varying in thickness from one and a half to three millimetres, and covering a cavity of a linear direction following the line of attachment above described. Its dimensions could (from the ragged nature of this part of the specimen) not be accurately ascertained; it was irregular, and three spurs from it are exposed in the cross-

section diagram accompanying. At its periphery the investing tissue merged gradually into the normal cortex of the neighboring gyri. The latter were puckered in toward the cicatrix, thus presenting a sort of radiating disposition of these gyri, in great contrast with the normal disposition of the left lobe. The right frontal lobe, as a whole, appeared smaller than its fellow.

"On making sections at brief intervals, it was ascertained that pathological appearances visible to the naked eye extended through the white matter, under the affected area, nearly to the frontal plane of the anterior end of the caudate nucleus. While the cortex of the anterior ends of all three frontal gyri, and in part the subcortical white matter, appeared of normal consistency and color, aside from the area immediately involved in the cicatrix, a large part—what may be called the 'core' of these three gyri—was converted into a spongy tissue, of the kind which is described by authors as resulting from rarefying encephalitis. Its centre was a finely meshed tissue, compressible and very nearly like a sponge in texture and feel. In the axis of the middle frontal the meshes appear wider, more honeycombed. The process did not appear equally advanced in all parts. The meshes were firmest, and the walls of the cavity most sharply defined, in the middle frontal, and least so in the orbital transition of the lower frontal, where there was an area of very recent lacunar softening. As a rule, the meshes appeared to follow the course of the nerve-fibres. The second section revealed a distinct fibrillation in the course of a projecting end of two arcuate fasciculi, comparable to that observed in fracture of the normal hardened brain."

Dr. Mendelson called attention to the appearance of the wound during an epileptic seizure, as described by Dr. Hume, as confirming the old theory of the causation of epilepsy formulated by Schroeder van der Kolk, namely, that the attacks are due to a spasm of the cerebral vessels. In this case both the absence of pulsation and the retraction of the skin covering the opening in the frontal bone clearly pointed to a diminution of the volume of the cranial contents, and the only conceivable way in which the cranial contents could, under the circumstances, be lessened would be by a diminution of the amount of fluid within the cranium, and the most obvious cause for this would lie in a spasm of the cerebral vessels. The case was also interesting from the location of the lesion in the frontal region—a region injury of which is not usually followed by signs of cortical irritation leading to epilepsy.

Dr. Hume said that the microscopical examination of the brain had not yet been completed, and it was possible

that a degenerative process would be found extending back and involving the motor tracts. Still the epilepsy might have been simply reflex in character occasioned by the irritation from the extensive lesion in the anterior lobe.

Dr. Eugene Hodenpyl presented specimens of

CARCINOMA OF THE STOMACH AND SUPRARENAL CAPSULES.

In the stomach the lesion was at the pyloric extremity. The regions where the suprarenal bodies should be found were occupied by two masses of friable tissue, and there were no evidences whatever of the proper substance of the adrenals. Examination under the microscope showed the new growths to be alveolar carcinomata.

Dr. Hodenpyl also showed sections of the suprarenal bodies from a case of

ADDISON'S DISEASE.

The patient was a woman, twenty-four years of age, and was born in Ceylon of English parents. Her father was living and healthy. The mother died at the age of seventy-three years. The family, on both sides, were pronounced brunettes, and the mother, especially, had an unusually dark complexion. It was often noticed that during periods of illness, even of a trivial nature, the patient's skin became much browner than at other times. There was no history of tuberculosis. During infancy and up to the age of fifteen years the patient had always been healthy. At this time she had a severe attack of sickness, lasting one month, the nature of which is obscure. The father states that the trouble began in "a gathering in the forefinger," followed by severe constitutional symptoms, delirium, fever, etc. About five years ago the parents noticed that the patient was failing in health. She began to lose flesh, strength, and appetite. She became stupid and apathetic. Her skin gradually became darker in color. She also developed attacks characterized by intense headache, nausea, and vomiting, diarrhœa alternating with constipation, great pain and tenderness over the abdomen, and occasionally delirium, lasting three or four days. These attacks varied a good deal as regards duration and severity, but they were more frequent during the past two years. The patient was admitted into the hospital suffering from one of her "bilious attacks." She was exceedingly dull and apathetic, and could give no good account of herself. On physical examination the skin was seen to be of a very dark brown color. The mucous membranes were considerably pigmented. Examination of the heart and lungs gave negative results. Considerable

pain and tenderness existed over the abdomen. The spleen was not enlarged. The urine contained a trace of albumin. Pulse, 90; respiration, 20; temperature, 100.2°. She had headache, nausea, vomiting, irregular bowels, and occasionally mild delirium. While in the hospital the patient's skin was at times of a decidedly browner hue than at others, and it varied from day to day, apparently without regard to her general condition. She finally passed into the "typhoid state," the stupor developing into coma, and she died April 29, 1888.

Autopsy.—The body was well nourished, the rigor mortis well marked. The skin over the whole body, especially the lower half, was of a dark, chocolate-brown color, the nipples and labia being almost black. The mucous membrane of the mouth, nose, and vagina was pigmented. The left lung was moderately œdematous, the right, firmly adherent to the chest-wall and diaphragm, congested and œdematous. In the apex there was a single small calcified nodule. The bronchial glands were not enlarged. The heart was small, flabby, and the muscle pale; the mitral valve was slightly thickened. The stomach was normal. The small intestines contained a number of congested and pigmented areas. The large intestine was normal. The liver was adherent to the surrounding parts, being attached by numerous dense and firm adhesions; the color was dark, and the cut surface smooth. The spleen was of normal size, very firmly adherent, soft, and contained three very small calcified nodules. The pancreas was normal. The suprarenals of both sides were enlarged to about three times their normal size. They were of stony hardness, and the cut surface showed that the medullary portion had been entirely converted into a cheesy and calcareous mass, the cortical portion being replaced by a pale grayish, semi-translucent material. The kidneys were rather large and firm, and of dark color, with the capsule adherent in places. The cortex was not thickened, and the markings were fairly distinct. The mesenteric glands were not enlarged. The thyroid was rather small and dense. The thymus was large, and projected as far as the third rib. Lying on either side and behind the trachea were two or three enlarged cervical lymph nodes which were entirely converted into cheesy and calcific material. The brain was normal.

Microscopical examination: Sections from the suprarenals and cervical lymph nodes, when stained for tubercle bacilli, gave negative results, although the lesion was evidently a tubercular one in an advanced stage. In and around the blood-vessels of the kidney were collections of small spheroidal cells, and scattered about were masses of small round cells between the tubules. The spleen

showed the lesions of congestion and hyperplasia. The liver was a little fatty. In the thyroid body, and principally around the periphery of the lobules, were here and there masses of small round cells, in places forming a zone of cells around a lobule; in other places they simply occurred as irregular masses. Moreover, there seemed to be an irregular infiltration of small cells into some of the lobules.

As regards the source of infection in this case, it is possible that the "gathering" in the finger might have been tubercular. The small round cells in the thyroid were very abundant. The speaker said that he had not made up his mind whether they were leucocytes or lymphoid cells. If they were the latter, the case would be an uncommon one, for lymphoid tissue was very seldom found in young people. In reply to a question whether tubercle bacilli had been found in Addison's disease, he said that Osler had discovered them in two cases, but that they are not ordinarily found in this disease.

Dr. J. C. Peters said that the first case of Addison's disease described in this country was presented at a meeting of this Society by Dr. John T. Metcalfe.

Dr. Henry N. Heineman, in response to a request to give a definition of the term "Addison's disease," after observing incidentally that Addison's disease seemed to be a much more common affection abroad than in this country, said that the affection is one in which certain phenomena, consisting in nervous symptoms and a pigmented skin, are associated with changes in the suprarenal bodies. These changes, however, are of the most varied character, and it is as yet impossible to give any exact definition of the disease, for the subject has not yet been sufficiently worked up to admit of such a definition.

Dr. John S. Ely presented two enormously enlarged

CYSTIC KIDNEYS

removed from a hog at the slaughter-house. There was hardly any kidney tissue remaining, the organs being made up almost entirely of cysts, which seemed to be retention-cysts. There was a very abundant growth of connective tissue.

Dr. Mendelson remarked that the specimens were interesting in connection with the question of gout in hogs. In a case of such gout described by him about a year ago, he had been able to show that this form bore a very close resemblance anatomically to the urate gout of man. The kidneys just presented showed that chronic interstitial nephritis—the "gouty kidney"—of human beings, one of the less obviously gouty lesions, was also represented in the hog. The speaker also dwelt upon the importance

of the study of comparative pathology, and urged members, whenever possible, to bring specimens illustrating diseases of the lower animals.

Dr. Mary A. Dixon Jones presented a specimen of

INTRAMURAL MYOMA OF THE UTERUS,

removed from a patient forty-four years old; married twenty-four years; no children. The tumor lay low in the pelvis, and made considerable pressure upon the bladder and rectum. It commenced at the internal os, completely occluding the uterine canal. There was also a subperitoneal fibroid, the size of an infant's head. Both tumors were removed by suprapubic hysterectomy, the stump removed per vaginam, as in kolpo-hysterectomy. The operation was successful, but the patient afterward succumbed to an old kidney trouble, complicated with heart disease. The microscopical examination of the uterine appendages showed long-existing disease.

A second specimen was one of

FIBRO-CYSTIC TUMOR OF THE BROAD LIGAMENT.

The patient was forty-eight years old; married twice; no children. Menstruation began at fourteen, accompanied, as she said, by terrific pain, which year by year became more and more severe. The patient had to have recourse to morphine. For years she was treated for uterine disease, without results. Probably disease of the ovaries commenced in early menstrual life. The growth had been observed for eight or nine years. When she came under observation in May, 1888, it was about nine inches in diameter, and the lower part was tightly wedged into the pelvis, pushing the uterus to the right. She entered the Woman's Hospital of Brooklyn May 30, 1888, and the tumor was removed three days later. It had an immense pedicle, with a great predominance of fibrous tissue. The patient made an excellent recovery. Microscopical examination of the tubes showed a very unusual condition, viz., hyperplasia of the mucosa, the mucosa being changed to fibrous connective tissue, a new formation of blood-vessels, and a new growth of epithelia.

Dr. Jones then exhibited a number of

MICROSCOPICAL SECTIONS OF DISEASED OVARIES.

Patient was forty years of age, feeble and emaciated. Nine years previously she had consulted an eminent specialist, who diagnosed her case inflammation of the ovaries. The disease continued to increase, the left ovary was changed to dense fibrous connective or cicatricial tissue, the outcome of chronic oöphoritis; some portions were calcified, containing globular deposits of lime salts,

and between them angular protoplasmic bodies like bone corpuscles. The right ovary was much enlarged, and most of it occupied by a fibroid growth or tumor, while in other portions of the ovary there were a number of small nodular fibromata, in the neighborhood of which the blood-vessels were much enlarged, probably by pressure. In the same ovary was a form of degeneration which Dr. Jones had not elsewhere seen described. In May, 1887, she presented to this Society a microscopical section, showing this formation. The patient had since emaciated and suffered much pain. Two years before, June, 1885, the speaker had removed diseased ovaries from a woman who had been an invalid for fifteen years. In these ovaries there was the same kind of formation. The case was published in *The Medical Record*, August, 1886, and the growth pronounced to be sarcoma. In August, 1887, she sent a microscopical specimen of this growth to Waldeyer, of Berlin. He replied October 18, 1887, that it was carcinoma. But from drawings made the summer previous, Dr. Jones could see that in the growth there were tracts of nucleated protoplasmic bodies, which nucleated bodies were coarsely granular, sometimes elongated, sometimes round, or forming an irregular network. When they were round and even they gave very much the appearance of round-cell sarcoma; but in some places these elongated protoplasmic bodies seemed to melt into each other, forming canals, in which were hæmatoblasts and the shadows of blood-corpuscles. Again, many of the protoplasmic bodies were like cut sections of blood-vessels, filled with blood-corpuscles, and in other portions there were completely formed blood-vessels. So the growth is a new formation of blood-vessels. But we do not know why an ovary should degenerate into this structure, or what is its pathological significance.

CANCER OF THE OVARY.

On March 28th, Dr. Jones reported to the Society the removal of a tumor from the floor of the pelvis, which was bound down by firm and strong adhesions, completely grown to the part. Before the operation the diagnosis was made of an enlarged left ovary, caused probably by an abscess. The patient had suffered for many years, but not more than many have suffered from apparently similar conditions. The removal was very difficult, but the patient made an excellent recovery from the operation, had a good appetite, and was apparently in excellent health. During the last two months, however, she has had an increase of pain, and an enlargement at the seat of the old tumor. A microscopical examination of the portion of the ovary that was adherent to the floor of the pelvis shows it to have been a cancer of the most malignant

type. The little connective tissue around the cancer nests were infiltrated with the cancer. The tubes were also infiltrated.

Dr. Henry P. Loomis presented a specimen of

CYLINDRICAL-CELLED EPITHELIOMA OF THE STOMACH, WITH EXCESSIVE DILATATION.

The patient, a male, forty-eight years of age, was admitted to Bellevue Hospital November 26th. His family history was unknown; he was grossly intemperate, had syphilis as a young man, and gave the evidences of tertiary manifestations. The patient could give no definite statement as to how long he had been ill, but thought he first began to vomit his food about a year ago. He said "he would go for several days eating and then gradually feel more and more loaded up, and finally vomit a quart or more." After unloading his stomach he would have a good appetite and would eat heartily again, but gradually his appetite would fail, and then he would feel an oppression in the epigastrium and would vomit. This was repeated constantly during the past year. Patient suffered a gnawing pain in his epigastrium for the past six months, which gradually became paroxysmal and has never become constant, he all the time growing weaker and losing flesh. Has never vomited blood, but only a slimy, greenish, sour-smelling fluid. During the past few weeks he failed rapidly; had complete anorexia during and after his attacks of vomiting. Bowels have been regular. During the whole of his illness he was anæmic and emaciated. Pulse rapid and feeble. Physical examination of the chest gave negative results. Liver dulness extended from the fifth rib to one and a-half inch below the free borders of the ribs. Abdomen very much retracted. Outline of the stomach was clearly defined, its upper border extended as high as the ensiform cartilage, while the lower border reached three inches below the umbilicus; the pyloric extremity of the stomach extended into the right lumbar region as far as the free border of the ribs; the cardiac end of the organ was in the left lumbar region. No tumor could be detected in the abdomen. The stomach was siphoned, and a large quantity of a slimy, black, sour-smelling fluid was withdrawn, and it was washed out with warm water and a pint of milk was then introduced.

The patient was comfortable for twenty-four hours, and then the washing-out had to be repeated. In spite of nutritive enemas and the introduction of milk into the stomach, the patient died within ten days from asthenia. The test for free hydrochloric acid was employed, with negative results.

Autopsy, made twenty-four hours after death: Lungs showed pulmonary œdema and congestion. Heart, normal. Liver, fatty infiltration with passive congestion. Kidneys, normal. Stomach measured eight inches along its lesser curvature, and twenty-three inches along its greater. It was greatly distended, and could contain about two and a-half quarts of fluid. The opening into the duodenum was so small that with difficulty a medium-sized probe could be passed. At its pyloric extremity was a tumor, circular in form and about an inch and a half in diameter, raised above the mucous membrane of the stomach less than a quarter of an inch. It was of dense consistency and with no portion of its surface eroded, and showed the microscopical elements of epithelioma. Intestines: all the small intestines were very much atrophied, and presented a pale, anæmic appearance.

The following are the interesting points of the case: There were none of the usual symptoms of cancer during life, for the case had been examined by a number of competent gentlemen who had not even suggested the possibility of cancer. No tumor was at any time detected, nor was the growth found at the autopsy sufficient to give signs of a tumor during life. The only symptom which suggested obstruction at the pylorus was the evidence of stomach dilatation. A microscopical examination showed it not to have been the ordinary variety of cancer met with at that situation.

Dr. W. P. Northrup presented a case of

EXTENSIVE INTERLOBULAR EMPHYSEMA,

with broncho-pneumonia, in a child aged five months. Patient was sick five days with signs of pneumonia; temperature 104° F. (rectum), and had a discharge from the nose two days before death.

There had been no whooping-cough, no eruptive disease before or with the last sickness, and no pseudo-membrane was visible. Had been accounted a strong and well child. On autopsy the body was found to be well nourished. A small abscess, located on the rib at its junction with the fourth costal cartilage of the right side, was discovered on separating the muscles from the bony wall. This was localized purely in the rib, affecting neither the pleura nor muscles. Pharynx contained moderate amount of mucopus, a thin pellicle of fibrino-pus, and was somewhat congested upon tonsils and elsewhere. Larynx had upon its surface a thin pellicle of fibrin and pus, which showed under the microscope reticulated fibrin containing in its meshes small round cells. This extended as far down as the bifurcation of the trachea. Right lung: broncho-

pneumonia of posterior portion of all lobes and extensive interlobular emphysema of upper; large air-sacs, of capacity of a drachm, wedge-shaped, from wide base at margin of the lung, growing smaller to the root. Left lung: broncho-pneumonia of the lower posterior portion. Other organs normal.

Dr. Northrup presented a case very similar to the present at the last meeting of the Society, and thought the second case would be of interest, taken in connection with the first. In neither case was there any convulsive muscular action to account for the lesion, nor any symptom of obstruction in the air-passages.

The Society then went into executive session.

Stated Meeting, December 26, 1888.

W. P. Northrup, M.D., President, in the Chair.

Dr. John S. Ely presented a monstrosity of the type

MICROMELUS.

There was no history of the parents of the child. The trunk and head were apparently well formed, but the arms and legs were almost rudimentary as regarded their length, although in thickness they were nearly normal. The bones of the arm and forearm were present, and the hands, though small, were not especially misshapen, nor were the fingers webbed. In the lower extremities the femur was very short and much curved. Both bones of the leg were apparently present, although it was difficult to assure one's self of this by reason of the small size of the member. The great toe stood out at nearly a right angle with the inner border of the foot, and the feet were inverted so that the soles nearly opposed each other. The appearance of the feet and great toes suggested the normal condition existing in apes.

Dr. L. Emmett Holt showed a series of specimens illustrating enlarged cheesy bronchial glands. The first specimen was one of

GENERAL TUBERCULOSIS, ENLARGED BRONCHIAL GLANDS, WITH ULCERATION INTO THE RIGHT BRONCHUS.

Female child, seven months old; family history unknown; three months before death she had an attack which was diagnosticated pleurisy, from which she never fully recovered. Had constant cough and considerable mucopurulent expectoration. There were continuous febrile symptoms for one month before death; the temperature range was between 101° and 104°, having the peculiarity that it was almost always higher in the morning than in the evening. There was progressive emaciation, moderate diarrhœa, no marked cerebral symptoms. There was much purulent expectoration. The physical signs gradually extended until there were signs of consolidation throughout the greater part of the right lung, in front and laterally, and scattered areas behind. Close to the spine, in the right interscapular region, there was a circumscribed area of marked dulness. The respiration here was exaggerated bronchial. Both were in striking contrast to the signs on the left side of the spine, and were interpreted to mean enlarged bronchial glands upon the right side. In the left lung there were no evidences of consolidation, but of a generalized bronchitis of the smaller tubes.

Autopsy, fourteen hours after death.—Brain: A few miliary tubercles were present, and a very slight amount of meningitis, chiefly over the right parietal lobe.

Chest—Right Lung: Upper lobe firmly adherent, and tears on removal. On section it shows many cheesy areas broken down in the centre, forming small cavities from one-eighth to one-half inch in diameter. One larger cavity near the anterior border filled with pus. The middle and lower lobes show many cheesy nodules, mostly smaller than in upper lobe. The intervening lung-tissue is in places consolidated and filled with miliary tubercles. Left lung: No adhesions; both lobes studded with small cheesy nodules, the longest being one-quarter inch in diameter, the intervening tissue being normal. Bronchial glands: They form a large mass above the right bronchus, the largest ones being nearly one inch in diameter; they are fused together. An ulceration exists in the right bronchus half an inch from the bifurcation of the trachea, which includes half the circumference of the bronchial wall. It communicates with a broken-down gland outside. Heart and pericardium normal.

Abdomen: The spleen was much enlarged (weight, seven ounces) and contained miliary tubercles. Many tubercles were found in the liver, also enlarged, and a few in the kidneys. The small intestine was healthy, except the ileum, which contained small tubercular ulcers in almost every Peyer's patch, and a very few elsewhere. The large intestine showed slight catarrhal changes only. Mesentric glands cheesy, the largest being half an inch in diameter. Stomach pale, otherwise normal.

A second specimen showed

GENERAL TUBERCULOSIS, TUBERCULAR MENINGITIS, CEREBRAL THROMBOSIS AND SOFTENING; ENLARGED BRONCHIAL GLANDS, WITH ULCERATION INTO THE RIGHT BRONCHUS.

Male, seven months old; family markedly tubercular upon the father's side; in fair condition until two months before death, when pertussis developed; attack rather more than average severity. Lost weight steadily, but had no other noteworthy symptoms till twelve days before death. His temperature was not high, only once touching 103°. A week before death the first signs of consolidation were found in the lungs; they were in the right upper lobe. Subsequently these signs were heard over the lower lobe and on the left side near the spine. At the last examination of the chest a circumscribed area of dulness was found in the right interscapular region. This was not accompanied by the peculiar respiration noted in the previous case, but by ordinary bronchial breathing rather

louder than usual. The diagnosis of enlarged bronchial glands was made, although no dulness was found on the upper part of the sternum. The spleen was easily felt below the ribs. Cerebral symptoms were present during the last week; first drowsiness, the next day opisthotonos and an attack of convulsions, and later, complete coma; pupils dilated, responding sluggishly; slight convergent strabismus; paralysis of the lower half of the right face; right arm and leg rigid; knee-jerk normal, or slightly increased; rapid pulse, and Cheyne-Stokes respiration. Convulsive twitchings were present during the last six hours. Death occurred quietly.

Autopsy, sixty hours after death.—Brain: Extensive basilar meningitis, more marked in the left Sylvian fissure. Lobes matted together by lymph, and pia studded with miliary tubercles. A few tubercles over the convexity. A firm thrombus in the left middle cerebral artery, not decolorized; a spot of red softening in the central portion of the frontal lobe, nearly as large as hen's egg; ventricles moderately distended with clear serum.

Cord not examined.

Chest—Right lung: Old adhesion over middle lobe quite firm, slight recent adhesions over lower lobe. Upper lobe shows a few miliary tubercles on its surface, and small cheesy nodules in the interior; middle lobe completely broken in its centre, forming a series of small cavities, filled with cheesy material and some fluid pus; lower lobe consolidated from broncho-pneumonia, with here and there minute cheesy spots at the lower portion. Left lung: A few miliary tubercles on the surface; on section, several small areas of broncho-pneumonia, and two or three broken-down cheesy nodules near the root. Bronchial glands: Very much enlarged on the right side, forming a mass two inches long and three-fourths of an inch wide. They are all cheesy, and some are broken down. There was an ulceration into the right bronchus, occupying exactly the site described in the previous case, viz., half an inch from the bifurcation of the trachea. This was nearly one-fourth of an inch in diameter. Glands on the left side were slightly swollen, but not cheesy. Heart and pericardium normal.

Abdomen: Spleen much enlarged, fully four times the normal size, filled with miliary tubercles, and many small cheesy nodules. Liver pale, tubercles on surface, and a few in its substance. Kidneys: A few tubercles on the surface; quite marked; cloudy swelling of cortex. Stomach normal. Small intestine normal, except ileum; Peyer's patches swollen and red, and nearly every one contains one or two small, circular tubercular ulcers. Large intestine: Half-a-dozen small ulcers scattered through the

colon. Mesenteric glands moderately enlarged and cheesy.

A third specimen illustrated

GENERAL TUBERCULOSIS; PERSISTENT OPISTHOTONOS, WITHOUT LESION OF BRAIN OR CORD.

Male child, aged seven months, always delicate; family history unknown. Weighed eight pounds on admission in July, being then two months old. During the next three months gained two pounds, and then gradually lost it. Emaciating slowly till death. In October attention was called to the child by reason of opisthotonos, which had then existed some weeks. The neck was so rigid that the body could be raised by the head. This continued with slight variation, but never being completely absent until death. An irregular fever began early in October, which lasted six weeks, the temperature ranging from 100° to 103° F. Careful and repeated examinations were made during this period, eliciting only a moderate enlargement of the spleen and the signs of general bronchitis over the chest, mainly of the larger tubes. There were no cerebral symptoms. Quinine was without avail. Moderate gastro-intestinal catarrh was present, but hardly enough to explain the symptoms.

In spite of numerous changes in diet, and finally wet-nursing, he lost ground steadily. Toward the last he had no fever, but little cough, and the opisthotonos, although present, was not so marked as in October. At the last recorded examination of the chest, two or three weeks before death, the only signs present were scattered coarse râles. He died of exhaustion December 18th, wasted to a skeleton.

Autopsy, ten hours after death.—Brain and cord absolutely normal; they were examined with great care. Lungs: Left, quite large areas of consolidation in both lobes, chiefly posteriorly; on section, many cheesy nodules and a few quite large areas infiltrated with cheesy products. Right lung, small cheesy nodules scattered through the whole lung; many miliary tubercles on the surface; very little pneumonia. Bronchial glands are all enlarged and cheesy. They form a large mass surrounding the trachea and bronchi, but lie chiefly in front. Most of the glands are the size of a hazel-nut, and are beginning to break down. Heart and pericardium normal. Spleen about twice the usual size; many small cheesy nodules and miliary tubercles. Liver congested; a few miliary tubercles. Kidneys: Quite marked cloudy swelling; no tubercles. Intestines: A few small, circular ulcers in the lower ileum, and about a dozen scattered through the colon; moderate catarrhal changes through

lower ileum and colon. Mesenteric glands size of a small marble, and cheesy. Stomach small, but mucous membrane normal.

Remarks.—In the first case the ulceration of the bronchus was probably produced by the breaking down of the gland exterior to it. The distribution of the inflammatory products suggested that this opening had existed for some time, and that the lesions in the middle and lower lobes had been set up by the aspiration of cheesy matter from the gland which had been discharged into the bronchus. The appearance in the left lung indicated this still more strongly; the matter having been coughed up into the trachea and then aspirated into the lung.

In the second case no cheesy gland existed near the opening into the bronchus, and this perforation was probably due to tubercular infiltration of the bronchial wall which had broken down.

In the third case the absence of all evidence of basilar meningitis at the autopsy was, in view of the persistent opisthotonos, rather surprising. The only explanation of the symptom which appeared was that it might have been reflex from irritation of some of the peripheral filaments of the spinal accessory by the tubercular gland which extended some distance into the neck.

Dr. Holt presented some additional specimens showing

DIPHTHERIA; MEMBRANE IN THE LARYNX, WITH NO CROUPY SYMPTOMS DURING LIFE; BRONCHO-PNEUMONIA.

A stout male child, aged sixteen months, was taken sick six days before death with high fever and sore throat. The temperature for the first twenty-four hours was 104° F.; during the next thirty-six hours it was low, ranging from 100° to 101° F.; during the last three days it ranged from 103° to 105° F. In the beginning both tonsils were swollen and congested, and a patch was seen upon the left. The next day one was noticed upon the right side also. There were no nasal symptoms and but moderate swelling of the cervical glands. The membrane in the throat did not spread beyond the tonsils. With the second rise of temperature the signs and symptoms of severe broncho-pneumonia developed which caused death. Throughout the attack there was no stupor, no laryngeal dyspnœa, and only slight hoarseness. Croup was not suspected.

Autopsy, twenty hours after death—Brain normal. Larynx: Epiglottis much swollen, especially at its borders, and is folded laterally upon itself. It is entirely covered with a thick adherent false membrane, excepting its base anteriorly. Thick membrane also covers the aryteno-epiglottidean folds. These are also swollen. The interior of the larynx is lined everywhere by a thin false mem-

brane which extends into the trachea nearly an inch. It is very loosely attached in the trachea, but quite firmly so in the larynx. There was moderate congestion and swelling of the mucous membrane of the trachea and larger bronchi, but no more false membrane encountered until the third or fourth divisions of the bronchi were reached. Here the membrane was abundant, but loosely attached; it could be drawn out of the small bronchi in pieces of considerable length. This was present only in the bronchi of the right lung, none could be seen upon the left side.

Both lungs showed extensive broncho-pneumonia; nearly the whole lower lobe and the posterior part of the upper lobe being involved on the right side, and quite a wide strip along the posterior border of both lobes upon the left side. The lungs were everywhere much congested. The heart showed quite large decolorized clots in both auricles and ventricles, larger upon the right side; they were but slightly adherent and probably post mortem. In the kidneys there was well-marked cloudy swelling of the cortex.

The other organs were substantially normal.

The President did not think that ulceration from the presence of enlarged glands was of very common occurrence. Referring to the case in which opisthotonos was present, he said that the causation of this symptom in young children was a subject of considerable interest. He had seen it in "marasmus babies" in which there was no brain or cord lesion discoverable post mortem. The only lesion constantly present, in the three or four cases which had come under his observation was gastro-enteritis. It was interesting also to note the occasional absence of symptoms in cases of glottic stenosis. He had that day removed from the body of a child a larynx in which there was a considerable amount of false membrane, as thick as blotting-paper, lining the opening in the larynx, and yet there had been no symptoms whatever of stenosis.

Dr. John C. Peters said that to this Society belonged the credit of first establishing the diagnosis of diphtheria as a distinct disease in this country. A number of specimens had been presented here during the years 1842, 1843, and 1844.

Dr. William P. Northrup presented some microscope slides of sections of specimens from three cases of

ACTINOMYCOSIS IN MAN.

Two of them were from two of the three cases operated on by Dr. Frederick Lange, of this city, and one was a specimen from Heidelberg. The ray fungus was well shown in one teazed unstained specimen, and differently

represented by a hardened section, doubly stained, in a second.

One was from a boy, from Pennsylvania, without history showing source of infection. The tumors were in the mediastinum.

The second was from a butcher of this city, who asked if his disease was like the "little pearls" seen in the lower jaw of cattle. The tumor, from which the sections had been prepared, lay at the side of the thyroid cartilage, and was about the size of a horse-chestnut. It communicated with the jaw and a carious tooth by a sinus, along which were scattered small, yellow, seedlike bodies, having the size and general appearance of miliary tubercules.

The infection entered through the cavity of the carious tooth, and advanced to the region afterward indicated by the tumor. Whether there were other similar tumors within the thorax, in the posterior mediastinum, was not indicated by symptoms at the time of operation.

The specimen was presented through the courtesy of Dr. Lange as a "card specimen," and will be fully recorded elsewhere.

The Society then went into executive session.

Anniversary Meeting, January 9, 1889.

WILLIAM P. NORTHRUP, M.D., PRESIDENT, IN THE CHAIR.

DR. E. C. SPITZKA presented

A MONSTROSITY ATTRIBUTED TO A MATERNAL IMPRESSION.

The specimen was sent to him for examination by Dr. William P. Lowman, of Orangeburg, S. C., who had been called to attend a colored woman, with the following history. When she was about two months advanced in pregnancy, while passing through some woods, she suddenly came upon a buzzard, who, having caught a pig, had devoured the greater part of its head. She was greatly startled, and was almost immediately attacked with uneasy sensations in the hypogastrium. Two weeks later these culminated in violent parturient pains. The doctor was called in at this juncture and learned the above facts. He diagnosed incipient miscarriage, which he succeeded in preventing. One month (twenty-eight days) after this severe pains set in again, and the contents of the uterus were expelled. Fortunately Dr. Lowman was called in, and his careful examination led to the discovery of a remarkable body, which on first sight struck him as resembling the body of a bird, about the size of a robin, but lacking the head. Even the skin resembled that of a plucked bird. The umbilical cord was normally attached, and the placenta—about two and one-half centimetres in diameter—also appeared normal. The feet presented a spread claw-like appearance, imitating a bird's foot even to the subsidiary posterior digit with its long claw.

The monstrosity was in every respect a maldeveloped human fœtus. The shape of the bones where exposed, the formation of the ribs, the minute characters of the articulations, and the disposition of the fœtal membranes, all left no doubt upon this head. Nevertheless, the surface resemblance to a decapitated and plucked bird was very great. The skin everywhere, except at the points of the extremities, presented the "goose-skin" appearance. The neck was long, bent over, ventro-lateral, and its terminal tissues appeared to be rounded off smoothly. The upper extremities terminated at the distal humeral end, which was covered by a membrane continuous with the subcutaneous tissues. The disposition of the thighs and legs was marvellously bird-like, particularly in the contour of the muscular masses. The feet were human feet, exhibiting a tendency to syndactylism; but on the

left foot was a claw, set back as in the bird's foot. The back of the monstrosity was prolonged into a caudal extremity very similar to what is seen in a bird. The rudimentary anterior extremities were so applied against the sides as to require no great stretch of the imagination to see in them the wing-bones. The chest also resembled almost exactly that of a robin.

Dr. Spitzka remarked that this case fulfilled one of the conditions demanded by the disbelievers in maternal impressions, namely, that the woman should have related her impression before the delivery of the fœtus. An interesting speculation might be indulged in regarding the unconscious psychological process and impression, which combined the impression of the *bird*, which had its head, with the *headless* character of the pig. It may be also questioned whether the—to a woman—familiar figure of a plucked bird was superadded. The case, the speaker said, offered a valuable and unimpeachable record of a maternal impression.

Dr. Walter Mendelson said that what struck him as very singular was that the monstrosity did not resemble the large bird that the woman had seen. Might the occurrence not be explained by assuming that the woman's mind was dwelling simply upon the appearance of a bird, and her thoughts then unconsciously took the direction of what was most familiar to her, the dressed barn-yard fowl?

Dr. Spitzka agreed with Dr. Mendelson in this explanation. It would be impossible, he said, for a maternal impression to produce a being of a different order, such as a bird with a beak or with feathers. In reply to a question, he said that he did not know how maternal impressions acted. But the cumulative evidence in support of the opinion that they were efficient factors in the production of monstrosities, in certain cases, was too strong to be gainsaid. It is not a valid argument against a fact to say that we do not know how it is produced. We see actual lesions occurring in hysterical patients as a result of purely psychological causes, yet we cannot trace the mechanism of their production.

Dr. Freeman presented the arm of an infant, four months old showing

ABSENCE OF THE RADIUS.

The first metacarpal bone and thumb were also wanting. Cases such as this, the speaker said, are not very rare, some thirty-five or forty having been reported. The history of the child's parents was unknown, and no cause for the deformity could be ascertained.

Dr. John S. Ely presented a heart showing the lesions of

MALIGNANT ENDOCARDITIS—INFECTION APPARENTLY FROM URETHRITIS.

Clinical History.—J. D——, male, aged twenty-eight, United States, waiter, single, walked into Roosevelt Hospital on March 17, 1888, in a very stupid condition. All that could be learned was that he had been sick for about three days. He was poorly nourished and anæmic; no sign of paralysis; pupils normal. Temperature, 105.8° F.; pulse, 130. During the night he was very restless and had to be tied down; vomited several times; passed urine and fæces involuntarily. The next morning his fever had abated somewhat, but his mental condition remained the same. On the 19th a partial left hemiplegia was noticed, and he frequently expectorated a brownish fluid. He failed rapidly, notwithstanding liberal stimulation, and died at 8.15 P.M.

Autopsy, by Dr. Delafield, nineteen hours after death. "Body fairly nourished. Brain, some increase of serum and some venous congestion. Old opacities of pia, more marked on left side. Brain normal. Lungs congested. Heart, old pericardial adhesions. Mitral valves have recent vegetations along margins. Aortic valves normal. Liver congested. Spleen, large and soft; contains some small infarctions. Kidneys, rather large, studded by a number of foci, apparently embolic. Stomach normal. Small intestine normal."

At the close of the autopsy, as no source of infection had been discovered, it occurred to Dr. Roosevelt, who was present, to examine the man's urethra, for any sign of inflammation. To his satisfaction, on squeezing the penis sufficient pus was obtained to make several cover-glass preparations and to inoculate some gelatine tubes. These, together with portions of the various organs, were sent to the Pathological Laboratory of the College of Physicians and Surgeons for examination.

Through lack of time the cultures were neglected and died before any observations were recorded, so that nothing determinate came of them. The cover-glasses and portions of the organs were, however, saved, and it is the result of their examination which I desire to report to-night.

Microscopical examination of the mitral valve shows, besides the old connective-tissue proliferation of chronic endocarditis, recent infiltration of the substance of the valve with small round-cells and fibrin, together with erosions on the surface of the valve, covered with fibrin and teeming with micro-organisms. These last are of two

forms, micrococci and bacilli. The micrococci are by far the more numerous and are arranged in clusters and in chains; both varieties retain the stain when Gram's method is employed. The bacilli are slender rods, about four times as long as broad. They are in very small numbers and stain by Gram.

In the liver there are occasional foci of broken-down tissue, each of which is surrounded by a zone of tissue densely infiltrated with small round-cells. In these small abscesses micrococci are readily to be seen when stained by Gram's method. Besides lying loose in groups and chains in the abscess-cavities, occasional masses of micrococci are occluding small blood-vessels surrounded by areas of more or less necrosed tissue. The liver shows also some congestion and fatty infiltration.

The splenic tissue is very soft and engorged with blood. Emboli of micrococci occlude many of the small arteries.

It is in the kidneys, however, that the most pronounced changes have occurred. The blood-vessels in many parts are much distended by blood. Large areas of degeneration of the epithelium of the tubules are present, and scattered here and there over the surface of the kidney are small abscesses, in the centre of which there are groups of micrococci. Emboli of micrococci are to be seen in the interlobular arteries, in the vasa recta, and in the vessels of the glomeruli. The micrococci are morphologically identical with those in the liver, spleen, and vegetations on the mitral valve. They stain by Gram's method.

The lesion in the urethra is of a catarrhal nature. The epithelium is swollen and detached from the basement membrane. In places, the desquamated epithelium is heaped up in considerable masses, and in these masses are small clusters of micrococci in clusters and chains, which stain by Gram's method and are similar, morphologically, to those found in the internal organs. None of them are present in the deeper parts of the penis, nor is any lesion in the corpus spongiosum apparent. No undoubted gonococci have been seen.

The pus obtained from the urethra at the autopsy is teeming with micro-organisms. A few of these resemble gonococci very closely, being small diplococci, flattened on the adjacent sides, occurring both in the desquamated epithelium and free, and staining with fuchsin. These are not visible in the specimens stained by Gram's method, and in this point correspond with Neisser's gonococcus. Large numbers of other micrococci are to be seen everywhere through the pus. They are in clusters and short chains, and stain by Gram's method. Measurement shows these micrococci to correspond closely in size

to those in the internal organs. They are not all of exactly the same size, but average about .9 micromillimetre in diameter.

As the result of the study of the specimens, the details of which have just been given, it is thought that the malignant endocarditis, with its attendant lesions in the other organs, may be attributed to the presence of staphylococcus pyogenes aureus and streptococcus pyogenes, and that it is not unlikely that these micro-organisms found entrance into the general system through the urethra.

The numerous and frequent complications and sequelæ of gonorrhœa have made it evident that the urethra may be an important site of inoculation of the general system. As far back as Lasègue's time the joint affection in gonorrhœal rheumatism was known to be a suppurative arthritis, and in 1880 Haslund [1] further directed attention to the marked differences between articular rheumatism and the gonorrhœal complication. The discovery of the gonococcus, and the fact of its presence in the pus of gonorrhœal joints, determined by Petrone,[2] Kammerer,[3] Smirnoff,[4] and Hartley,[5] remove all doubt as to the nature of the trouble and the source of the infection. This is a striking example of infection of the system from the urethra, but others are equally so: for instance, complicating epididymitis and orchitis, and suppurating buboes; abscesses in the prostate and Cowper's glands.

That a non gonorrhœal urethritis occasionally occurs is coming to be quite generally recognized. These cases are usually of short duration, and gonococci are absent in the discharge. Bockhart [6] has described a micrococcus, differing in many respects from the gonococcus, as occurring in this form of urethritis.

Again, the possibility of *mixed* infection has been attracting attention, and there are now a number of cases on record of this nature. Bumm [7] found the pyogenic bacteria to be the most frequent cause of these mixed infections. That such cases should occur is not strange,

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[1] Haslund, A.: Gonorrhoisk Rheumatisme og Pyarthrus gonorrhoicus. Ugeskr. f. Lägev.. 4 R. I., No. 1, pp. 1–10; No. 2, pp. 17–26.

[2] Petrone, L. M.: Sulla natura parasitaria dell' artrite blenorragica. Rivista clinica di Bologna, 3 ser., iii., No. 2, pp. 94–113.

[3] Kammerer: Ueber gonorrhoische Gelenkentzündung-Centralbl. f. Chir., 1884, No. 4.

[4] Smirnoff: Etiology of Gonorrhœal Arthritis. Lancet, August 28, 1886: Review.

[5] Hartley, F.: Gonorrhœal Rheumatism, Especially in the Female. N. Y. Med. Journal, vol. xlv., 1887, p. 376.

[6] Bockhart, M.: Ueber die pseudo-gonorrhoische Entzündung der Harnröhre und des Nebenhodens. Monatshefte f. prakt. Dermatol., v., 1886, No. 4.

[7] Bumm, E.: Ueber gonorrhoische Mischinfectionen beim Weibe. Deutsche med. Wochenschr., 1887, No. 49, p. 1057.

considering that Lustgarten and Mannaberg [8] have described eleven varieties of micro-organisms as occurring in the normal urethra, and among them staphylococcus pyogenes aureus. Bockhart [9] details two cases of such mixed infection; in one there was a suppurating bubo, in the other a peri-urethral abscess. In both cases gonococci were absent from the pus of the abscess-cavities, and staphylococcus pyogenes aureus was present in large numbers.

The literature of malignant endocarditis and pyæmia is not devoid of cases in which the source of infection seemed to lie in the urethra. In a somewhat hasty review of the literature since 1880, I have come upon the following: In that year Schedler [10] reported a case of malignant endocarditis following gonorrhœa, in which gonorrhœal joints complicated and, seven months later, were thought to be the source of infection to the heart. The patient died about a month after the involvement of the heart. In 1881 Weigert [11] called attention to the frequency of lesion of the male genitals in pyæmia; of 17 cases of pyæmia examined, 8 had lesion of the genitals (abscess of the prostate, vesiculæ seminales, etc.). He does not, however, allude to the lesion in the genitals as the source of the systemic infection. The same year a case of pyæmia secondary to gonorrhœa was described by Holst.[12] In this case, also, gonorrhœal joint trouble was present. Two years later (1883) Petersen [13] published an interesting résumé of 975 cases of gonorrhœa treated at the Alexander Hospital in St. Petersburg. Of these 537 were complicated cases, in two of which the complication was peri-urethritis, with pyæmia as the cause of death in one of these. In 1884 Dérignac [14] cited a case of endocarditis secondary to gonorrhœal joint affection. This case recovered. In Paget's [15] investigation (1886) of 500 cases of pyæmia, 97 were thought to depend upon lesion of the genito-urinary organs. As Paget does not

[8] Lustgarten and Mannaberg: Ueber die Mikroorganismen der normalen männlichen Urethra und des normalen Harnes, mit Bemerkungen ueber Mikroorganismen im Harne bei Morbus Brightii acutus. Vierteljahrschr. f. Dermatol. u. Syphilis, xiv., 1887, p. 905.

[9] Bockhart, M.: Ueber secundäre Infection (Mischinfection) bei Harnröhrentripper. Monatshefte für prakt. Dermatol., 1887, No. 19.

[10] Schedler, Paul: Zur Casuistik der Herzaffectionen nach Tripper. Inaugural Dissertation, Berlin, 1880.

[11] Weigert, C.: Zur Technik der Microscopischen Bacterien-Untersuchungen. Virchow's Archiv, lxxxiv., 1881, p. 275.

[12] Holst, J. C.: Tilfälde af Gonorrhœ med. Pyæmi. Norsk. Mag. f. Läg., 1881, 3 H., x., 389.

[13] Petersen, O.: Seltene Complicationen bei Urethritis. St. Petersburg med. Wochenschr., 1883, No. 45.

[14] Dérignac: Endocardite blenorrhagique. Gaz. Méd. de Paris, 1884, No. 7.

[15] Paget, Stephen: The Distribution of Pyæmic Abscesses. Lancet, 1886, ii, 203.

cite the cases in detail it is impossible to learn how many of them were dependent upon gonorrhœa. Unquestionably two of them were thought to be, for on consulting an article by Savory in "St. Bartholomew's Hospital Reports," vol. iii. (1867), to which Paget refers as the source of 133 of his cases, I find these two attributed to gonorrhœa alone. Besançon [16] in 1886 reported two cases of malignant endocarditis in which lesions in the genito-urinary organs, which originated from the urethra, were thought to be the source of general infection. In the first of these, old suppuration of the seminal duct and epididymitis were found at the autopsy; in the second, abscess at the neck of the bladder, behind old strictures. In both cases pyogenic bacteria were obtained from the various lesions. In 1887 Lancereaux [17] added two cases to the list. In both malignant endocarditis was secondary to disease of the genital organs; in the one case of the prostate, in the other of the testicle. In the same year Van der Velden [18] described two cases of acute endocarditis secondary to gonorrhœa. Although both lived, there was thought to be no doubt as to the relation of the two affections. Recently Roswell Park [19] has reported a case of pyæmia thought to have resulted from gonorrhœa. The etiology of the case is somewhat obscured because of an old abscess at the sterno-clavicular articulation.

There are, then, on record at least nine cases of malignant disease, be they classified as cases of pyæmia or of malignant endocarditis, in which the urethra appears to have been the point of entrance of the micro-organisms. In none of these cases, so far as I am able to learn, were any cultures made, and, in so far, the proof of the etiological relation between the two affections is lacking. The morphological identity of the micro-organisms, in the cases examined, in the lesions of the genital organs and in those of the internal organs, certainly points to a connection between the two. It is somewhat difficult to conceive of a urethritis as resulting from a malignant endocarditis, and that the two lesions should be mere coincidences, without closer relation, in so many cases, though perfectly conceivable, seems to me improbable. In our case the fact that, though carefully looked for, no other point of infection was found, strengthens the theory that the urethritis was the primary lesion. It is a pity that no

[16] Besançon, M.: Endocardite ulcéreuse à point de départ génital chez l'homme. L'Union Méd., 1886, Nos. 100 and 101.

[17] Lancereaux: Endocardite à point de départ genital chez l'homme. L'Union Méd., 1886, No. 100.

[18] V. d. Velden, R.: Zwei Fälle von "Endocardite blenorrhagique." Münch. med. Wochenschr., 1887, No. 11.

[19] Park, Roswell: Pyæmia as a Direct Sequel of Gonorrhœa Journal of Cut. and Gen.-Urin. Diseases, vi., 1888, No. 12.

statement of the duration of the urethritis could be obtained *ante mortem*, as much light might have been thrown upon the case by it.

Our knowledge of the etiological relation between urethritis and malignant endocarditis and pyæmia may be summarized as follows:

1. Pyæmia has been shown by the investigations of Rosenbach [20] and Passet [21] to be due to the presence of the pyogenic bacteria—staphylococcus pyogenes aureus, albus, and citreus, and streptococcus pyogenes.

2. Micro-organisms belonging to this same class have been found in the valvular lesion of malignant endocarditis by Wyssokowitsch,[22] Weichselbaum,[23] Ziegler,[24] Prudden,[25] and others, and experimental endocarditis in animals has been produced by the above-mentioned writers and by Ribbert,[26] by the inoculation of the same micro-organisms.

3. The complications of gonorrhœa are very numerous, and for their explanation the possibility of introduction of micro-organisms into the general system must be presupposed.

4. In certain cases of mixed gonorrhœal infection of the urethra, pyogenic bacteria have been found along with the gonococci (Bumm,[7] Bockhart [6]).

5. Nine cases are on record in which malignant general infection occurred in patients suffering from gonorrhœa, and in some of these (Besançon,[16] Lancereaux [17]) micro-organisms morphologically identical with the pyogenic bacteria have been found in the urethral discharge as well as in the lesions in the internal organs. In these nine cases the urethra was the only discoverable point of infection.

The case which has been reported is thought to be another of the same nature. Whether the coincidence of the urethritis and the general disease be merely accidental, or whether the one bears an etiological relation to the other, is the question. There is certainly no inherent impossibility in the idea that infection should find entrance to the system through the urethra. Why should not ulcerated urethral mucous membrane offer as good op-

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[20] Rosenbach: Mikroorganismen b. d. Wundinfections-krankheiten des Menschen. Wiesbaden, 1884.

[21] Passet: Untersuchungen u. d. Aetiologie der eitrigen Phlegmone des Menschen. Berlin, 1885.

[22] Wyssokowitsch, W.: Beiträge zur Lehre von der Endocarditis. Virchow's Archiv, ciii., 1886, 301.

[23] Weichselbaum, A.: Zur Aetiologie der acuten Endocarditis. Wiener med. Wochenschr., 1885, No. 41.

[24] Ziegler, E.: Lehrbuch der speciellen pathologischen Anatomie. Fünfte Aufl., 1887, p. 39.

[25] Prudden, P. M.: An Experimental Study of Mycotic, or Malignant Ulcerative Endocarditis. American Journ. Med. Sci., January, 1887.

[26] Ribbert: Ueber experimentalle Myo- und Endocarditis. Fortschr. d. Med., 1886, No. 1, p. 1.

portunity for the entrance of micro-organisms as ulcerated mucous membrane in other parts of the body? It remains only for some intelligent investigator to prove it in a few cases by a careful study of the micro-organisms found in the varicus lesions in pure cultures.

Dr. W. P. Northrup presented some specimens illustrating

HEMORRHAGE INTO THE SUPRARENAL BODIES, WITH DISCOLORATION OF THE SKIN,

incomplete septum ventriculorum, and sprue. A female child was admitted to the New York Foundling Asylum when one day old and was entered upon the books: "Condition poor—conjunctivitis—occasional cyanosis." There was a superficial ulcer over the soft palate extending upon the hard, which on the following day was pronounced diphtheritic. Two days before death the temperature reached 103° F., and dulness developed over the left lung, behind. A distinct exudate was seen upon the tonsils. The child died suddenly, on the eighth day of life, of heart failure, thought to be due to diphtheritic poison. Beyond occasional cyanosis there is no further note of its color during life. On autopsy the body was fairly nourished, the skin and subcutaneous tissue having a yellowish-brown or dirty yellow color; skin slightly mottled. The color was distinct and not to be overlooked. The trachea and bronchi contained a moderate amount of tenacious, dirty, frothy mucus; the mucous membrane was not congested and there was no exudate upon its surface. Upon the walls of the pharynx, the soft palate, and extending down the entire œsophagus, was an abundant growth of sprue or thrush (which had been called diphtheria during life). There was no ulceration, and the pellicle differed from the fibrinous exudate by appearing coarsely granular. This was best seen upon the œsophagus of the specimen. The stomach contained an increased amount of tenacious, dirty mucus and gave no evidence of the presence of any vegetable growth. The jejunum and ileum contained dark-greenish masses of mucus in a dirty, frothy fluid. Follicles were not swollen. The bronchial glands were slightly enlarged. Left pleural cavity contained about ℥ jss. of a dirty brown fluid, in which were shreds of fibrin. The visceral pleura of the lower two-thirds was fringed with fibrin and overlay a densely solidified lung. Of the right lung but a small portion of the lower posterior portion was affected by congestion. The pleura and pericardium contained a small amount of dirty brown fluid.

INCOMPLETE SEPTUM VENTRICULORUM.

There was a direct communication between the ventricles of the heart, by an opening at the attachment of the aorta, to the left base and the valve-attachment of the right tricuspid.

The communication was through an interval left in the development of the fibrous auriculo-ventricular bands. The opening was circular, 6 mm. in diameter, looking into the left ventricle at the attachment of the aorta to the ventricle, its upper convex margin interdigitating with the lower convex attachments of two semilunar valves.

It looked into the right ventricle, having, upon its left, the inner portion of the ventricular wall, which was at this point prolonged into the infundibulum or conus arteriosus. Above and to the left it had the ventricle muscle. It had below and to the right the fibrous attachments of the tricuspid valves, opening behind the cusp, which had its attachment nearest the pulmonary orifice.

During systole, then, the tricuspid valves being closed, the blood would tend to escape behind the cusp, through the opening, in the manner of the coronary circulation behind the aortic valves. On the other hand, the contraction of the left ventricle would tend to equalize or overcome this pressure, until some peripheral embarrassment in circulation should disturb the equilibrium, when cyanosis would appear. Of such temporary disturbance the history makes mention. The foramen ovale was patent, in its posterior portion, about the normal degree. Ductus arteriosus was also pervious.

Kidneys were congested and contained urates in the papillæ which radiated from apex to base in reddish, fine lines. The suprarenal gland of the right side was increased in thickness, its middle third being dark and congested. The left gland was three centimetres long by rather more than one in thickness, pyriform in shape. The color was mostly opaque white, bordered on one side of its greatest enlargement by an irregular zone of recent hemorrhage. The gross appearance was that of cheesy degeneration of tubercle. Microscopic examination showed a small amount of normal gland-tissue in the smaller end of the pear-shaped body, a large mass in the middle of broken-down tissue in degeneration giving the opaque white color already referred to; beyond this there were signs of recent hemorrhage. In the midst of the broken-down tissue was a large vessel containing a thrombus. There was no tubercle tissue; the destruction of the gland was due to hemorrhage. In the congested middle zone of the right gland were numerous bilirubin crystals which showed distinctly in the specimen presented.

The anatomical diagnosis of the case is twofold. First, it is apoplexy of one suprarenal body and pigmented skin, in a child of eight days. It is not considered a case of Addison's disease. Apoplexy of the suprarenals is not among children regarded as rare by writers. Congestion and discoloration of the medullary substance is common, but, in Dr. Northrup's experience, apoplexy, with destruction of tissue and enlargement of the body, can be said to be rare. Second, the case is one of sprue or thrush, which was diagnosticated diphtheria, and malformation of the heart, with sudden death. There were patches on the tonsils and palate, which appeared to be pseudo-membrane to a man eminently capable of judging; and when the child died, suddenly, it was thought to be heart failure after diphtheria.

The microscope showed the fine, granular, grayish pellicle to be oidium albicans, growing upon the surface of the mucous membrane.

Dr. L. Emmett Holt said that he had recently come across the statement that oidium albicans was not the fungus concerned in the formation of sprue, but that it was saccharomyces albicans, and he asked whether this was a fact, which had been affirmed also by others.

Dr. Mendelson said that when in Leipsic, in 1882, he remembered having heard the statement that the sprue fungus was not oidium albicans, but a mycoderma.

Dr. Holt asked whether Dr. Northrup had ever noted the presence of the thrush fungus in the intestinal canal.

Dr. Northrup replied that he had never seen it below the œsophagus.

The Society then went into executive session.

The following officers were elected for the year 1889:

President: William P. Northrup.
Vice-President: H. M. Biggs.
Secretary: Thomas L. Stedman.
Treasurer: J. H. Hinton.
Editor: J. C. Peters.

Committee on Admissions and Ethics: J. C. Peters, L. H. Sayre, H. P. Loomis, Walter Mendelson, and L. Emmett Holt.

Committee on Publication: G. C. Freeborn, Walter James.

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ERRATA.

Page 65, seventh line from top, "Fluctuating" should be "Functionating."

Middleton Goldsmith Lecture

BY

J. M. DA COSTA, M.D., LL.D.

PROFESSOR OF THE PRACTICE OF MEDICINE IN THE JEFFERSON MEDICAL COLLEGE, OF PHILADELPHIA

THE RELATIONS OF DISEASES OF THE KIDNEYS, ESPECIALLY THE BRIGHT'S DISEASES, TO DISEASES OF THE HEART.

ON THE RELATION OF THE DISEASES OF THE KIDNEY, ESPECIALLY THE BRIGHT'S DISEASES, TO THE DISEASES OF THE HEART.

Lecture I.

When, by your kind thought, I was asked to deliver the Middleton Goldsmith lecture before your learned body, several subjects occurred to me which, from their great pathological interest, seemed worthy of the occasion, and which I believe the generous founder of these lectures would have approved of, could his cheery presence be here to animate us. From among these I choose, partly because it has been with me one alike of pathological and clinical investigation, the relation of diseases of the kidney, especially the Bright's diseases, to diseases of the heart. Much has been done of late years to examine this subject; but it is far from being clear or complete, and I must claim your indulgence if, in the endeavor to add a little to our knowledge of it, I allude to some facts which to you must be trite.

We all know that there is a close connection between diseases of the kidneys and diseases of the heart. But how intimate is this connection, and what determines it? Is there anything in an affection of the heart which would lead to a kidney affection? Or is the kidney disorder the starting-point of the undoubted combination observed? Again, are the lesions, when found together, both due to a common cause which determines them, and not the direct consequence of each other?

Allow me to detain you with a brief discussion of these weighty questions, before I call your attention to some of the finer points of pathological research which I shall attempt to demonstrate to you.

One of the first questions to solve is, Whether disease of the heart, in itself, leads to disease of the kidneys? The general impression is that it often, indeed generally, does. But on analyzing a large number of cases I find that this belief is incorrect. I will here give some of the results of my investigations into the matter, and will take the records of the Pennsylvania Hospital for a period of ten years, extending from the beginning of 1877 to the beginning of 1887, and similar records from the Clinical

Department of the Jefferson Medical College for a period from June, 1875, to January 1, 1888, selecting merely the cases of which it was reasonably certain that the cardiac affection was the primary lesion.

At the Pennsylvania Hospital were noted of such cases of *valvular heart affections*, 76; at the Jefferson Medical College Clinic, of valvular heart affections, 51. Of these 127 cases, in all of which the urine was examined, for the most part repeatedly and at different times, and in many of which, especially in the cases at the Pennsylvania Hospital, autopsies are recorded, there were in 92, it is specifically stated, no evidence of any kidney affection, as shown by the absence of casts, of albumen, and, in a number of instances, by the notes of the post-mortem changes. The 76 valvular heart cases at the Pennsylvania Hospital furnished 28 cases in which some concurrent kidney affection was observed; the 51 cases at the Jefferson Medical College Clinic showed but 7. Of these 7, in 1 (Case III. of series), happening in a child with a mitral regurgitation, there had been scarlet fever three years, and an attack of rheumatism two years prior to the record of the case; there was hypertrophy with dilatation in connection with the valvular disease, and chronic parenchymatous nephritis. In another case (Case XIII.), that of a man fifty-two years of age, the mitral lesion, following rheumatism, was associated with urine one-fourth albuminous, and of character usual in parenchymatous nephritis. The dropsy began in the abdomen, the swelling there preceding that of the feet and legs for nearly a year. In the third case (Case XXV.), a presystolic mitral murmur was found with hypertrophy of moderate degree, but with considerable dilatation. The heart's action was irregular, the urine was full of albumen, and attacks of cardiac asthma and occasional uræmic convulsions occurred. In the fourth case (Case XX.), a married woman, twenty years of age, there was some abdominal dropsy and a puffy face. The valvular lesion consisted in aortic regurgitation with a slight mitral regurgitation; considerable hypertrophy of the heart existed. The pulse was 128. The urine was free from sugar, of specific gravity 1.018, and contained small amounts of albumen. The kidney was considered to be in a state of parenchymatous inflammation.

Of these 4 cases, in all of which, probably, the kidneys were in a state of chronic parenchymatous nephritis, the first ought really to be excluded, since the kidney affection much more likely had its origin in scarlet fever, leaving thus only 3 undoubted cases.

In 3 more of the series albumen is mentioned as occurring in very small quantities. In one (Case XVI.), it was

present without tube casts; in another (Case XXXI.), where a mitral presystolic murmur as well as an adherent pericardium existed, a few hyaline tube casts were discerned in a feebly acid urine, free from bile and sugar, but containing a small amount of albumen and crystals of oxalate of lime. In a third, in which mitral regurgitation with considerable hypertrophy and dilatation was noted, albumen in small and varying amounts was detected, but nothing else. In all three instances it was due to congestion of the kidneys, and not an evidence of Bright's disease. We have then but 3 cases in 51 in which a kidney affection really existed.

I will now examine the 28 cases out of the 76 recorded at the Pennsylvania Hospital, in which the urine examinations indicated coexisting disorder of the kidneys. In 16 of the 28 the record speaks specifically of but small amounts or of traces of albumen and of the absence of casts. In one of these (Case I.), the autopsy tells of kidneys showing no marked alteration of structure except that, weighing respectively six and seven ounces, the capsules were thickened, and the right kidney contained two small cysts. The highest amount of albumen recorded in these 16 cases is one-sixth of the test-tube, sometimes one-tenth, and often the merest trace.

In 6 cases (Cases II., XVI., LV., LXIII., LXIX., LXXVI.), tube casts are described in addition to the albumen. They were hyaline or slightly granular casts. In 2 of these 6 cases we have autopsies; and this is a summary of the record. In one (Case LXIII.), the hypertrophied heart weighed thirty-six ounces, there was marked insufficiency from contraction of the aortic leaflets, slight thickening of the mitral; the kidneys were congested, cyanotic, otherwise normal. In the other (Case LXIX.), the heart weighed fifteen ounces; the mitral and aortic valves were both thickened. The kidneys were congested, but not altered in structure.

In 6 cases the clinical record is that of much more serious disease. But one of these (Case LXV.) we must at once exclude, as the albumen, the pus, the epithelium, which existed in connection with the mitral disease was clearly of vesical origin, and any renal implication was most doubtful. Five cases of marked renal affection, with highly albuminous urine, we still have to account for. Of these, in one (Case LXI.), there were also pus cells and bladder epithelium; but distinct renal casts were detected, and the case was looked upon as one of parenchymatous nephritis complicating mitral insufficiency. In two (Cases XLII. and LIV.), the same view was taken of the renal complications, associated in Case XLII. with mitral narrowing; in Case LIV. with both mitral and

aortic insufficiency. But in this case, in which highly granular and hyaline casts abounded, and in which the albumen was in bulk two-thirds of the test-tube, the conclusion is vitiated by the patient having been a great drunkard, and all his tissues having been profoundly altered. Case XLVII. is very instructive on account of its clear history. It happened in a man who had rheumatism thirteen years prior to admission into the hospital, but in whom dropsy and puffiness of the face only appeared two months before. The urine contained albumen, granules, and fatty casts. The greatly enlarged heart weighed, with clots, thirty-six ounces; without clots, thirty-two ounces. The mitral orifice readily admitted four fingers, so did the tricuspid orifice; the aorta was fully twice as thick as normal, but the aortic valves were competent. The walls of the right auricle were double the natural thickness; the left ventricle, its cavity enlarged about one-half, had walls measuring near the base one inch. The kidneys were engorged, and appeared in the early stages of parenchymatous nephritis. The left kidney, weighing twenty-four ounces, contained a few cysts on the surface; its blood-vessels were prominent, but not thickened. The right kidney was about the same in size and structure.

In Case LXX. we also have an autopsy. The aortic and mitral leaflets were thickened and contracted. The kidneys were large, firm, harder than normal, and evidently diseased, but showed no adherent capsules or signs of contraction.

Summing up the whole inquiry, we find in 127 cases of valvular disease of the heart but 8 in which any true affection of the kidney existed—anything more than mere congestion. And among these 8, there is not one instance of the contracted or cirrhotic kidney. In truth, the kidney disorder that results from a valvular disease of the heart is simply a congested kidney of full size, redder, a little more glistening, and in elderly people, or where the congestion has been of long standing, firmer than normal, showing even slight increase of fibroid texture, and, perhaps, a rather more adherent capsule. Persistent engorgement of the renal veins gives the well-known livid, cyanotic appearance to the somewhat indurated organ. The epithelium of the tubes is swollen, and finally undergoes granular degeneration.

I have, by the analysis of the cases, endeavored to give you a proof how rarely valvular disease of the heart leads to chronic disease of the kidneys, other than congestion. When it does, the form of affection is that of a chronic congestion, which, in its turn, passes into a parenchymatous nephritis. The granular contracted kidney does not, I believe, result from the process of congestion just alluded

to ; though, as I have already said, occasionally, especially in elderly people, the kidneys from long-standing congestion may appear hard, and exhibit a slight amount of fibroid change. These statements seem at variance with those of one of the most eminent of your members, Dr. Delafield,[1] who, in 137 cases of death from heart disease, reports 27 large white kidneys, 29 atrophied kidneys, and 28 of chronic nephritis which could not be classed as either large white, or atrophied organs. Perhaps the apparent discrepancy is to be explained by the cases being heart disease of which it was not specifically known whether it preceded the renal affection or followed it.

During life it may be very difficult in individual cases to determine whether we have, as the result of the heart malady, simply a congestive condition of the kidneys, or real Bright's disease ; in truth, as in both states dropsy is a prominent symptom, we obtain but little aid from the general symptoms, having to base our opinion largely on an accurate cardiac investigation. It may be thought that the urine would afford us much assistance ; but this it does not always do. A good many of my observations, in purely cardiac cases, speak of urine of low specific gravity, scanty, containing traces of albumen, and here and there casts. The character of these casts is, for the most part, epithelial and not highly granular, though this, too, must not be taken as an absolute rule. On the whole, the most important diagnostic sign, with reference to the urine, is found in the slight and varying amounts of albumen and in the infrequency of the casts. Moreover, though it has been stated that the urine, in even these heart cases with renal complication which is not Bright's disease, may have a low specific gravity—in one of my cases it was only 1.006—as a rule, the specific gravity of the scanty urine is high, ranging from 1.020 to 1.024 ; in one instance of mitral disease my record speaks of 1.030. Sugar was examined for, but found only once (Case XXII. of the Jefferson Medical College series) ; here there was aortic regurgitation. Urates, my notes say, are often abundant ; biliary coloring matter is not infrequent.

The character of the pulse, the absence of marked arterial tension, and the precise physical signs of the cardiac disease, give us more valuable diagnostic evidence, taken as a whole, than the state of the urine. We may also lay stress on the want of uræmic symptoms, and avail ourselves of the ophthalmoscope as a means of diagnosis. It has, in fact, happened to me several times to determine, by finding albuminuric retinitis, the true meaning of the renal change and the secondary nature of the valvular af-

[1] System of Medicine by American Authors, vol. iv.

fection. In one of these instances, indeed, albumen was at several examinations absent, tube casts were but few, the specific gravity was, as a rule, low; but the whole progress of the case, the uræmic symptoms which arose, the absence of dropsy, showed, irrespective of the eye appearances, that the renal malady was the preponderating and original affection.

Before proceeding, it may not be without interest to point out that the most common form of valvular disease, associated with renal disorder of the kinds we have found to exist, is mitral narrowing. In 8 of the cases at the Pennsylvania Hospital it is noted only in 2 that the urine is free from albumen. In the cases in which a real structural affection, a parenchymatous nephritis, is developed, mitral stenosis has a preponderating influence. Next stands—for tricuspid disease is so rare that I have not data sufficient to judge by—mitral regurgitation. In uncomplicated aortic disease albumen and casts are very seldom detected in the urine; so seldom, that it is always a question whether a mitral complication is present when kidney engorgement is found.

Pure *hypertrophy* and pure *dilatation*, instances, therefore, of enlargement without valve affection, give us the same form of kidney derangement we have been studying. But with reference to hypertrophy, unless the cavities are at the same time decidedly stretched we do not find albuminous urine. Thus this existed in only 1 out of 10 cases noted at the Jefferson Clinic. In pure dilatation, urine albuminous in traces, indicative of congestion of the kidney, is more common. Still, it does not exist in the majority of cases; and I have known it not to appear until the last week of life, even though that life had been a burden for months, owing to the enormous dropsical swelling and the turgid lungs.

We have been studying diseases of the heart and the kind of kidney affections they induce, selecting for analysis cases where, from the history, or the post-mortem results, or from both, the record of the antecedent malady was clear. We now turn to diseases of the kidney and their combination with diseases of the heart. It is well known that this combination is a frequent one. We find diseases of the kidney associated with valvular disease of the heart, with hypertrophy, with dilatation, and with pericardial affections. In studying the subject I mean to draw conclusions only from instances in which, from the history as well as from the concomitant features of the case, we may fairly judge that the kidneys were the first, or, at least, simultaneously, diseased.

Let us examine the valvular diseases of the heart as they are found associated with affections of the kidney.

I find, looking at the records of the Pennsylvania Hospital for ten years, from the beginning of 1877 to the beginning of 1887, that in 101 cases of renal disease in which the state of the heart is particularly mentioned, 41 cases have a concurrent valvular heart affection, and in 60 there is no such lesion. Of the total 101 cases, 57 are acute Bright's disease and 44 chronic. Of the 41 renal cases with valvular lesion of the heart, 29 are Bright's cases of the chronic type, and 12 are of the acute type. Of the 29 cases of chronic Bright's disease with associated valvular heart lesion, it is noticed that 13 have a preceding history of rheumatism. In 5 there is a positive statement of no preceding rheumatic history. In the other 11 cases nothing is remarked about rheumatism.

Of the 12 acute cases with concurrent valvular lesion, 3 cases have the record that rheumatism did not precede. In the other 9 cases nothing is said with reference to a rheumatic history.

Out of 29 cases of chronic Bright's disease with valvular disease, the age of the youngest was thirty-three years; 14 of the 29 were beyond the age of forty-five years; the oldest was sixty-five years of age.

Of the 44 chronic renal cases, 8 were examples of the large, white kidney. The ages of these 8 were from forty-eight to sixty years. One case had double mitral and aortic murmurs, and was preceded by several attacks of acute rheumatism. Of the other 7, nothing is stated about either the heart or a rheumatic history.

Of the 57 acute renal cases, in none are mentioned signs of cardiac hypertrophy without a valvular affection, nor, indeed, do the notes show that even with this complication was the heart enlarged.

In the notes of the Clinic of the Jefferson Medical College, in 21 acute cases of Bright's disease, 20 of which had marked dropsical symptoms, the heart was found to be normal in all but 4, 2 of which had had a previous history of acute rheumatism. In these 4 cases there were evidences of valvular disease without hypertrophy.

From an analysis of these observations it becomes evident that the character of the kidney affection, in combination with the valvular lesion in the heart, is, in the vast majority of instances, the contracted kidney. Next stands acute Bright's disease, where, also, we find valvular affections, but here without the hypertrophy so common in the first group. In the acute, the strong influence rheumatism exerts is very evident; nor is that influence lost in the chronic, where an unusually large proportion are associated with rheumatism—too large a proportion for us to assume that it is a mere coincidence.

Let us inquire what the state of the valve lesion is. It

is chiefly an affection of the mitral valve, consisting in a thickening of the mitral, with here and there rough deposits. Next in frequency, but with similar pathological changes, we observe a lesion of the aortic valves, occasioning either aortic narrowing or aortic regurgitation. A further cause of the valve affection is seen in some of these cases to be due, I believe, to the hypertrophy of the heart forming part of the Bright's disease preceding some general thickening of the valve, though even this may be wanting. A stretching of the valve subsequently takes place, but insufficient to close the orifice in the enlarging heart. Thus the hypertrophy and the dilated condition of the cavities give rise to the valvular imperfection, rather than this to the enlargement of the heart. It is in this way that I explain some very striking and interesting cases of hypertrophy occurring in advance of the valvular lesion, which I have observed, and in which the autopsies showed simply this imperfect closure of the orifice with a slightly thickened mitral valve, and at times with shortened and more rigid papillary muscles.

I have known, but far less often, a similar state at the aortic orifice. The aortic disease that existed was due to the stretching of the valve, which, having reached a certain point, could go no further. This stretching, with the simultaneous thickening, produced an opening through which the blood flowed back. It is thus that we may have a valvular disease following hypertrophy with dilatation, rather as the result of this process, than the hypertrophy and dilatation as the result of it. If you ask me whether this is a common cause of valvular lesion in Bright's disease, I say it is distinctly not; it is very much less common than marked valvular thickening with degenerative change.

You see, then, that the valvular disease of the heart which occurs in diseases of the kidney may be of varying origin; chronic thickening, deposits, degenerative changes, coexisting rheumatic alterations, which have led to these changes, and, lastly, mere hypertrophy and dilatation, which, associated, perhaps, with slight valve thickening, or with rigid papillary muscles, in its turn leads to valvular imperfection. Omitting the last-named group, the ordinary valvular affections in the Bright's diseases are the result of the altered tissue nutrition of the valves and the degenerative changes which take place there, as they take place in both the large and small vessels of the body. They are favored to a greater or less degree by the morbid products which, from want of proper elimination on the part of the kidneys, circulate in the blood. It may also be a question whether, in part at least, the altered nutrition of the valve may not be due to affection of their nervous supply. The changes, the degenerative ones

certainly, are favored by age. Yet we cannot assume that age is the important factor it appears at first sight; for the acute Bright's affections show also, as has been proved, a strong tendency to the implication of the valvular apparatus, and the acute affections are much more common in the young, or, at least, in younger people, than are the chronic affections. Let me further notice, once more, a point brought out in the analysis of the cases submitted to you, namely, the frequent existence of rheumatism. This may well produce the valve injuries at the same time that it leads to other changes in the body; and thus be a general cause, both for the valvular affection and for the disease of the kidney and of other textures.

Before ceasing to examine the aspect of the subject, I will call your attention to some purely clinical facts which must make us receive with caution many of the statements of coexisting valvular disease of the heart and of Bright's disease. It is often recorded that there was present a soft murmur, variously heard; in some instances at the apex, in others at the right base. Now these murmurs in Bright's disease are not the result of valvular affection. They come from the state of the blood; or they are simply murmurs from temporary perversion of valve action—what we might call functional murmurs. They are inconstant, they are soft, and they are not signs of valvular disease of the heart. You will easily see that the error is all the more readily committed because we have hypertrophy, with or without dilatation, as such a common attendant upon Bright's malady. Thus, the mere hearing of a murmur in the heart is not a sign of valve affection in Bright's disease, not even when there is coexisting hypertrophy. We have to take into account its persistence, its character, generally rough, the state of the second sound of the heart, and the fact that it may be heard in the axilla, and posteriorly near the angle of the scapula, to make clear that it is of organic type, and not of the temporary character just referred to. Bearing this in mind, we shall have to reject a great many of the cases of so-called valvular disease of the heart, often loosely reported, in which the only evidence of the valvular affection is based upon a murmur, the particular character of which is not stated; or its softness or temporary nature is alluded to, but an erroneous conclusion is drawn from its existence.

The difficulty does not exist with reference to diastolic murmurs, which, in my experience, are always organic. But it is not lessened by a study of the pulse, which is in itself altered by the arterial lesions of the Bright's malady and by the thickened left ventricle; nor can we place,

for similar reasons, much dependence on sphygmographic tracings.

I think, then, that the number of cases of valvular disease of the heart, existing as a complication of Bright's disease, is more often overstated than understated. Indeed, with the greatest care, unless the case has been for a long time under observation, it may be almost impossible to detect the true relation between the maladies. Much of the difficulty will be, however, solved, if we accustom ourselves to look upon them as not dependent upon each other, but due to a common cause, working almost simultaneously, or even at different periods—mischief in kidney, heart, and other textures. To the frequent starting of this common cause in rheumatism, attention has been called.

Let us pass to an examination of the *hypertrophy of the heart* which exists in combination with Bright's disease of the kidneys; I mean where the hypertrophy, with or without some dilatation, is a pure process, and not complicated with valvular disease. With reference to the acute cases of Bright's disease, I have already mentioned that we do not find hypertrophy. In the 22 cases of this character at the Jefferson Clinic, and in the 57 cases at the Pennsylvania Hospital—therefore in 79 cases—in most of which there was marked dropsy, in not one were the signs those of hypertrophy, not even in the instances with valvular lesion. A few doubtful exceptions are observed in the notes where strength of impulse is mentioned; but in these it is probable that the valvular affection which coexisted had its origin in a previous attack of rheumatism.

Thus, then, we may assume as positive that hypertrophy of the heart does not occur in acute Bright's disease. This is vastly different in the chronic form of the malady. Here, as is well known, hypertrophy is the rule. This subject is a matter of such common observation that it scarcely needs re-enforcement by figures; but I will quote, since they are based solely upon post-mortem records on a most extensive scale, the recent researches of Goodhart in "Guy's Hospital Reports" for 1886.

In the ten years from 1873 to 1882, inclusive, there were autopsies of 344 cases of granular kidney, and of 196 of chronic parenchymatous nephritis. The weight of the heart in the cases in which the kidneys were described as granular was in only 103 under twelve ounces, which was, therefore, within the limits of average weight. In 226 it was above this weight. Making allowance for all possible exceptions, as old age and wasting disease, and adding 36, which were hypertrophied hearts, though of average weight, there are 262 cases of marked hypertrophy in 329 undoubtedly granular kidneys.

After making all deductions for the cases of chronic parenchymatous nephritis, there were in 25 only a heart of average weight; in 119 there were hypertrophy of the left ventricle, a proportion of over one to four. In lardaceous disease of the kidney, hypertrophic changes in the heart and arteries were not observed, which agrees well with the general observation on the subject.

Dilatation of the heart becomes associated in a more or less marked degree with the growing muscle. It affects pre-eminently the left side of the heart. In Goodhart's paper, just quoted, I find 51 cases of notable dilatation of the left ventricle associated with hypertrophy in the 344 cases of granular kidney, and 33 cases in the 196 cases of chronic parenchymatous nephritis. It would therefore appear that the dilatation predominates in chronic parenchymatous nephritis, while pure hypertrophy does so in granular kidney.

In cases of hypertrophy, such as I have described, the heart attains very great size; the muscles are firm; for the most part it is a pure hypertrophy, only here and there there takes place some increase of the fibrous texture, as well as of the muscular. The muscular fibres are a little more opaque, and sometimes more granular than normal. As the disease advances, secondary degeneration, granular and fatty, may occur; but they do not happen unless there be decided coexisting disease of the vessels.

The state of the vessels in these instances of hypertrophy is very interesting to note. They show the same condition which has been found in the kidneys, in the brain, in the spinal marrow, in the lungs, and in many other parts of the body.

The inner coat is thickened, the muscular coat hypertrophied, the outer coat in a state of fibroid change, and the whole lumen of the vessel greatly lessened by these encroaching lesions. This lessening of the calibre is the same, as are the changes the same that we find in the renal vessels, and it may take place to a very great degree. My colleague at the Pennsylvania Hospital, Dr. Arthur V. Meigs, has recently shown me a section of a heart, obtained from a man under his care who died of granular kidney, with aortic valve disease, and left ventricle from three-fourths of an inch to an inch thick, exhibiting besides increased muscle beginning interstitial change. Microscopically examined, the first ascending branch of the left coronary artery was so much thickened, chiefly by the increase of the intima, that the lumen of the vessel was a mere chink, the thickening being irregular, about four times more on one side than the other.

You will readily see how such a state of things might lead to secondary degeneration by cutting off the blood

supply from the hypertrophied muscle; and it is most likely that these degenerations which we have in the hypertrophied hearts of Bright's disease, which, however, I believe, are not the rule, come on as an after-result of the altered vascular supply.

As a few minutes ago I called your attention to some points of diagnosis with reference to valvular disease in Bright's affection, I may now call your attention to some points with reference to hypertrophy. It is not always easy to make this out clinically. We have to consider not only the size of the percussion dulness, but the extent of the impulse, as well as its force and position; the lowered apex-beat, which is often felt on a line with or beyond the nipple; and the heavy character of the first sound. Nor must we fail to take into account the character of the second sound, its accentuation at the base, which commonly happens in these hypertrophies in connection with renal change. Strong, extended impulse, displaced apex, altered sounds, and increased percussion dulness are the phenomena by which we judge; but not by any one of these alone, certainly not by the impulse, which may be temporarily increased by other causes than cardiac hypertrophy. On the whole, I lay most stress on the altered character of the sounds of the heart, both at the apex and the base, and the changed position of the impulse.

The enlargement of the heart which takes place in Bright's disease of the kidney may be made use of in prognosis. We are often greatly at a loss to form an opinion as to the probable length of the case and to the way in which it is progressing. Up to a certain point the occurrence of dropsy and of eye changes will assist us, as well as the evidences of marked arterial tension. But dropsy, while common in chronic parenchymatous nephritis, is very generally absent, or but slight, in cirrhotic kidney. The urinary examination does not always afford us much light, for some of the worst cases are those in which—I allude especially to the cirrhotic kidney—the amount of albumen is small, or often temporarily absent, and in which the tube-casts are few; nor can we lay too much stress on the character of these casts. It is under these circumstances that I think the heart condition becomes an especially valuable means of prognosis. Where the heart is but slightly affected there will be reason to believe that the case will continue for some time, provided we do not observe signs of uræmic poisoning. Where, on the other hand, the hypertrophy is marked, there is ground for the fear that the disease is more general, is advancing, and that the case will not belong to those long cases of the affection which we sometimes encounter. Thus the study

of the heart may be made use of as a means of estimating the amount of general change and the likelihood of shorter or longer life.

Let me reiterate the view that, according to my experience, the hypertrophy in the majority of instances is a pure hypertrophy, unconnected with degenerative changes. It is certainly so in the earlier stages of the affection. In instances of long-standing disease and in elderly people these degenerative changes may be found in the progress of the case; but my observations do not at all agree with those of Buhl, who has stated that fibrous and chronic interstitial myocarditis is very frequently found in connection with the hypertrophy. To its occasional occurrence I have already alluded.

Dilatation of the heart, unconnected with hypertrophy, or at least predominating over the increase of the muscular walls, is extremely rare. When it happens, it is the latter kind of cases, namely, beginning as hypertrophy, but the rate of increase of the muscular structure not remaining in proportion to the dilatation of the cavities that is met with. Among the cases at the Jefferson Clinic I have collected some instances of what might be fairly called dilatation unaccompanied by hypertrophy, or attended only by a slight degree of hypertrophy; and I have seen a small number in private practice. Goodhart points out that in the young the risk of dilatation of the heart is much greater than in older persons.

Another lesion of the heart which we observe in connection with Bright's disease, both acute and chronic, is *pericarditis*. But inflammation of the outer membrane of the heart forms only part of that general tendency to serous inflammation which we find in Bright's disease. It is probably due to the contaminated state of the blood, and neither its occurrence nor its explanation need here detain us.

Of all the heart affections passed in review, hypertrophy is the most common. Let us look at its cause. I leave out of consideration those cases where clearly existing valvular disease may have led to the hypertrophy; though even here it is always questionable whether some of the increased growth be not due to other causes than the valvular affection, and may not have preceded it. I will take the pure instances of hypertrophy and ask, How are we to explain them? It will serve our purpose best if, in considering this much mooted and most intricate subject, we give a short summary of the different views held as to the cause of the hypertrophy, rather than attempt an historical review of how and by whom these views have been brought forward.

One of the main views entertained is, that the hyper-

trophy of the heart is due to the blood contaminated by renal excrementitious matter, exerting a stimulating effect upon the action of the heart, thus leading to hypertrophy of the ventricle; and, further, that the impure blood requires greater force of ventricular contraction to propel it through the unwilling vessels. This view has the great authority of Bright, and is to this day advocated by some eminent teachers.

A second view, while taking note of the influence of the impure blood, holds that the altered blood tension produced by the disease of the kidney, and the difficulty of the blood circulating through it, give rise not only to the well-known feature of heightened arterial tension, but to increased size of the heart to overcome the resistance of the renal organs. This is largely the view of Traube and some of the German investigators, and has been recently reaffirmed by Cornil.[1]

A third view is that the left ventrical beats with augmented force because the arterioles refuse to receive the altered blood, and in consequence of the resistance these vessels hypertrophy, especially in the arterial coat. To overcome this muscular resistance, the heart has to act with greater force, and its muscles increase. This is, in the main, the view of George Johnson, and is largely based on that change in the muscular coat of the arteries which he has so well described.

A fourth view is that a fibroid degeneration, a fibroid overgrowth of the outer coats of the arteries, takes place over a large portion of the vascular system. In consequence of this, the heart has to struggle to overcome the resistance of the permanently narrowed vessels, narrowed by these hyaline, fibroid changes; and the enhanced blood-pressure shows not only in the raised arterial tension, but the increased resistance leads also to cardiac hypertrophy. This is mainly the view of arterio-capillary changes, of a vascular fibrosis as the cause of hypertrophy of the heart, which has been strongly and ably advocated by Gull and Sutton.

The last two views have much in common, at least as regards the production of the hypertrophy in consequence of the altered condition of the vessels.

I will mention a fifth view, which for the present I shall simply indicate, namely, that the hypertrophy, as well as to a great extent the vascular changes, are the result of a common process which takes its origin in the ganglionic nervous system. This is the view which in its main features flows out of some researches made by Dr. Longstreth and myself, and which here I simply state, in-

[1] Compte-Rendu du Congrès International, tome i. Copenhagen, 1886.

tending presently to call your attention to the particular facts bearing on it.

Now, analyzing these different views, let us first take notice of the assumed influence of impure blood, and it is for practical purposes possible at the same time to examine into the supposed alteration of the blood-pressure due to the resisting kidneys. These views which I thus group together seem to me untenable. If true, why should we not have hypertrophy of the heart, and especially the form of hypertrophy far the most common, of the left ventricle, in other diseases of the kidneys than Bright's disease? Why should not, too, in the acute form of this malady, the heart be increased? It might be objected to this argument that, in the acute affection, the disease does not last long enough to produce muscular increase; but the objection will not hold good with reference to the other diseases of the kidney, in which serious, we might even say destructive, kidney lesions take place, in which uræmic symptoms happen, yet in which there is no cardiac hypertrophy. I will take the instances, a number of which have come under my observation, of persons dying from extensive cystic degeneration of the kidneys, in whom the heart was found absolutely normal.

Here is one. In a gentleman whom I attended, with a very large tumor in the abdomen, and who passed bloody and albuminous urine, there were for three weeks before death most marked uræmic symptoms. At the autopsy both kidneys were enormously enlarged, measured each about fourteen inches in length, were filled with cysts, and no trace of renal structure could be detected. It was, indeed, difficult to understand how life could have lasted so long with such extreme disorganization; yet the heart was absolutely healthy.

In a second case, the kidneys measured six inches in length, nearly four in width, and three and a half in thickness. The tissue was almost wholly converted into cysts, from the size of an egg to that of a pin's head. There was no disease of the heart. This specimen is now in the Museum of the Pennsylvania Hospital.

In a third case the two kidneys were greatly enlarged, irregular in outline, the capsule was roughened by shreds of inflammatory adhesions; the organs, measuring each about seven inches in length and four in width, were filled with cysts. The thoracic viscera were in an absolutely normal condition. The liver was found to be highly fatty. The kidneys are also in the Museum of the Pennsylvania Hospital.

I shall cite two more cases, partly to sustain my view, and partly on account of the evident uræmia which preceded death. A man, under forty years of age, by pro-

fession a gambler, was admitted into the Pennsylvania Hospital in a comatose condition. He had been struck at by a companion, while drinking in a saloon, but was not hit. Immediately afterward, to the astonishment of those around him, he fell to the ground unconscious, and remained in this condition until death, a day or two later. No history of his previous state of health was obtainable, further than that he had seemed well, and was nightly occupied at his business as a dealer in gambling games. The urine was found to contain some albumen, and a large amount of sediment, mostly of a cellular character. Death occurred in the hospital with all the signs of marked uræmia. The urinary secretion was extremely scanty, and the unconsciousness profound.

The circumstances connected with the death led to the arrest of the companion who aimed the blow; but the investigation of the coroner showed that death was due to natural causes, and not to violence. At the autopsy, the kidneys were found to be in an advanced state of cystic degeneration. They were greatly enlarged in all directions, and the tissue was almost entirely converted into cysts. The disease had evidently been of long duration. The lungs were normal, so was the heart.

Yet another case. A young servant-woman, who had been in good health so far as known, fell on the floor while engaged at the wash-tub, and was found lying in an unconscious condition. She partly regained consciousness, but, on her admission into the hospital, could give no satisfactory history. She died a few days later in a comatose state, with every evidence of uræmia. The kidneys showed advanced cystic degeneration. The heart was normal, and no lesions were presented in the other organs, save congestion of the lungs.

I have thus presented to you five cases leading to death with the most advanced cystic degeneration of the kidneys existing, with the blood evidently impure, and, finally, occasioning, in the majority, marked uræmic symptoms, yet with actually normal condition of the heart.

I might go further in this line of argument, and call your attention to the enormous kidneys with calculi embedded in them, kidneys which produce abdominal tumors large enough to be mistaken for gastric and omental cancers, also leading to death, and in which no disease of the heart was found; so, too, in instances of cancer of the organ; though, in this condition certainly, and even in cystic degeneration at times, the argument is somewhat vitiated by the fact that, while one kidney undergoes extreme change and disorganization, the other may remain normal, or, at least, be still capable of performing its function fairly well.

The next views, those of arterial changes, whether in the muscular coat producing muscular hypertrophy, or from fibroid growth in the outer coats, I shall not discuss, further than to say that I believe the anatomical researches on which they are founded to be correct, having often seen the conditions referred to. Nor is it necessary to go into the question which is the most common of these changes; for I think that both may be found in different cases, and even in the same case at different stages. While thus concurring in the correctness of these observations, I will point out how insufficient they are to interpret the cardiac hypertrophy; how impossible it is to prove that blood is resisted by the altered vessels on account of its character; how difficult it is to explain the enlarged heart simply as the result of vascular changes, when we have striking alterations in the vessels, both large and small, in old age, and from syphilis, and in and beyond aneurisms, where we do not find cardiac hypertrophy happening as rapidly, or to anything like the extent that it does in Bright's disease, if, indeed, it happen at all.

Let me now, before taking up the fifth view, which I told you had grown out of observations by Dr. Longstreth and myself on the nervous ganglia, call your attention briefly to these observations, and supplement them by some which have not hitherto been published. Let me add, and here acknowledge, that in the researches about to be brought forward, which were most difficult and laborious to carry out, it is to the care and attention of Dr. Longstreth that they owe their merit, rather than to anything I have myself been able to do. Let me also recall that in the investigations we made on Bright's disease[1] we found marked changes in the renal ganglia, of a character showing increased fibrous tissue and atrophy of the ganglion cells. These were associated with decided alterations in the renal vessels, as exhibited to you in these micro-photographs, which will make the matter clearer to you than I can from description. This condition in the renal ganglia has been confirmed by Saundby,[2] who, however he differs with our conclusions, has seen exactly the same pathological lesions.

I shall proceed to present to you the statement based upon 11 cases accurately observed, in which the cervical ganglia from which the cardiac nerves arise were minutely examined; and 5 of these cases, which tell the tale of all, I will give you in some detail. The records relate particularly to the inferior cervical ganglia, giving off mainly the inferior cardiac nerve. All of the cases were

[1] American Journal of the Medical Sciences, July, 1880.
[2] British Medical Journal, January 13, 1883.

interstitial nephritis; 9 were chronic contracting kidneys, and 2 contracting kidneys of more acute form.

Case I.—Emma H——, aged sixty-five, admitted into the Pennsylvania Hospital May 18, 1885; died May 18, 1885; admitted for cerebral hemorrhage; had vomited; was unconscious, and completely relaxed or paralyzed; had a few irregular convulsive movements; two quarts of highly albuminous urine were drawn by catheter; she died in three hours. Post-mortem examination showed an extravasation of blood in the external part of the left hemisphere, but it had not extended to the ventricles. Death resulted in this case apparently from uræmia, rather than from the cerebral clot directly. The kidneys were very much reduced in size, with thickened capsules and granular surfaces, dotted with small cysts. The left ventricular wall was much thickened, measuring over one inch. The minute structures of the inferior cervical ganglion showed many marked changes; the ganglion cells were deeply pigmented, appeared shrivelled or compressed; equally to be noticed was the tissue around the ganglion cells, composed of a delicate fibrillar tissue in which appeared very numerous round and spindle nuclei. Some of these nuclei were doubtless those of the nerve structures, but the majority were a new growth of connective and fibrillar elements; in places they were so numerous as to obscure the ganglion cells. In the greater part the vesicular nucleus of the ganglion cells could be seen, but the nucleus was generally visible, faintly stained with carmine. A very striking feature was the appearance of the vascular structures; in the first place, the capillaries seemed in some parts to pervade nearly the whole area, some with a narrow calibre carrying but a single red blood-corpuscle, and from this width upward to those with six corpuscles abreast, and many of these vascular tubes presented ampullar dilatations; some of this condition can be accounted for by the mode of death, but the most was due to permanent changes. The walls of the arterioles showed the thickening so often found and described in the arterial walls in these cases of renal disease.

Case II.—Hannah G——, aged seventy, admitted September 4, 1884. No. 920. Died November 13, 1884. Had been gradually failing in health, and lately had chest symptoms and œdema; the secretion of urine had slowly diminished. On admission there was orthopnœa, the lungs were full of coarse râles, and were emphysematous, rendering any accurate determination of the size and condition of the heart impossible. The apex of the heart was far to the left of the normal. There was ascites, and also diarrhœa.

The heart showed hypertrophy of the left ventricle, its

tissue was undergoing fatty changes and atrophy; the kidneys, although much altered, did not exhibit the marked changes of the other cases. The minute structure of the inferior cervical ganglion cells indicated atrophic conditions; the cells were pale, with less than the normal amount of pigment (as usually seen in the ganglia of adults dying of disease), the nucleus was plainly visible, but the nucleolus stained with carmine very faintly. Between the ganglion cells the tissue was of firmer character than normal, and appeared to encircle these cells with stiff fibres. There were no round nuclei, but only flattened spindles in this space. The capillaries were not conspicuous, but the walls of the arterioles were altered, though to a less marked degree.

Case III.—Mary McG——, aged fifty-two, admitted November 3, 1884. No. 1,288. Died January 30, 1885. Her history tells of a severe cold, followed by swelling of the legs; urine of dark color and much reduced in amount; later, nausea and vomiting. On admission, the urine was smoky and contained fifty per cent. of albumen; the microscope showed blood, hyaline, and granular casts, and also some epithelial casts. Only twelve ounces of urine were passed in twenty-four hours. Dyspnœa and diarrhœa existed. Later came uræmic convulsions and strongly urinous odor of the breath and body. Post-mortem examination: Heart weighed sixteen ounces; right distended, left contracted and much hypertrophied. Liver small, pale, and granular. Kidneys reduced in size, firm, capsule adherent, and the surface granular. The case was an acute attack of nephritis in a contracting kidney.

The microscopic examination of sections of the kidney of this case was very interesting, showing the majority of the Malpighian bodies converted into firm fibrous nodules, and only in a few places were the convoluted tubes to be seen. Here and there a Malpighian body presented somewhat of a normal aspect, with a few tubes around it, in which the epithelium could be distinguished. The chief feature was the fresh outpouring of small indifferent cells, as seen in acute interstitial nephritis. The ganglion cells were in an advanced state of degeneration, full of granules, shrivelled, and the nucleus and nucleolus scarcely to be distinguished. Between these cells the tissue presented a fibrous aspect.

Case IV.—Rose B——, aged thirty-seven, admitted December 13, 1884. No. 1517. Died January 12, 1885. The attack followed gradually after pregnancy, with greatly diminished urine secretion—one-half teacupful in twenty-four hours. Diarrhœa and swelling of abdomen were noted. The lungs were full of râles, and the pleural cavities contained effusion. The heart-action was rapid, the

pulse "corded," the artery hard. The urine was albuminous and contained granular casts. Post-mortem examination: Pleural and abdominal effusions; old pericarditis; hypertrophy of the left ventricle, the wall measuring one and a half inch; liver showed cicatrices, possibly syphilitic; kidneys very small, the capsule thickened, the surface highly granular, the cortex had almost disappeared, while the cones were much distorted.

The inferior cervical ganglion presented even more highly altered conditions than in the previous case. In not a few instances the cells seemed to have nearly vanished, or a few grains of pigment evidenced their site. Other cells, while showing some pigment, looked blurred, shrivelled, and irregular in outline. The nucleus was misshapen, and the nucleolus was like a fragment of poorly stained matter, pushed over to one side of the nucleus and attached to its wall. Around the cells the fibrous tissue formed circles, closely resembling the thickening of Bowman's capsules in a contracting kidney. Even the spindle nuclei in the tissue between the cells had lost their distinctness, and the fibres looked less firm and rigid, but coarser.

Case V. — Mary H——, aged thirty-four, admitted January 4, 1885. No. 1647. Died January 8, 1885. For four months had had œdema, and for two days orthopnœa. The urine contained one-sixth albumen, and hyaline and granular casts. Death was sudden. Post-mortem examination showed old pericardial thickenings; weight, twenty ounces; the left ventricle measured one and one-half inch. The kidneys weighed each one and one-half ounce, capsule thickened, and tightly adherent; the surface was very granular. On section, the cortex had almost disappeared, and the pyramids were much distorted.

This specimen was less well prepared for examination than the others, owing to the delay in obtaining the autopsy; but however much the ganglion cells changed through the delay, this factor cannot account for the alterations of the tissue of the cardiac ganglion. The general conditions were the same, very nearly, as in the previous specimen.

I will not present to you any more details of cases, but will rather exhibit to you some microscopical drawings, taken directly from sections. Fig. 1 shows the ganglion cells treated with the ordinary carmine staining, highly granular and pigmented; their outlines indistinct; the nucleated nucleus scarcely visible. The texture between the ganglion cells is crowded with small, rounded, or spindle-shaped nuclei. These nuclei are specially numerous around and overlying the ganglion cells. The speci-

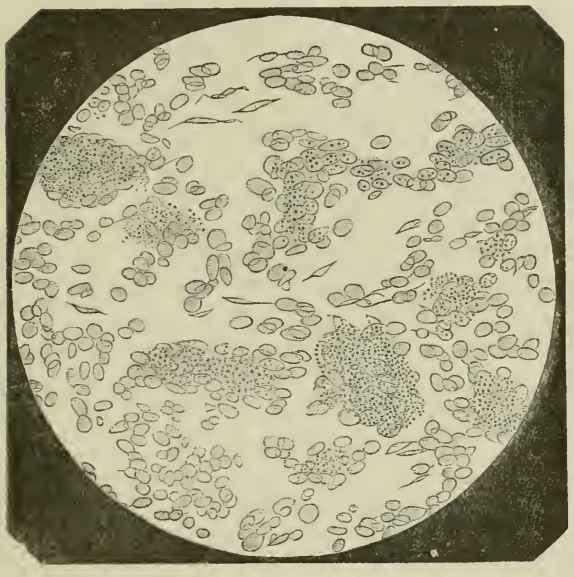

FIG. 1.

FIG. 2.

men is from a case in a moderately advanced stage of degeneration. Fig. 2 shows the lesion in a later stage. The whole tissue is firm and hard looking. The ganglion cells are surrounded by rings of coarse fibres, in which are a few remaining spindle nuclei. The ganglion cell itself has greatly shrivelled; the nucleus is pressed to one side, the nucleolus showing indistinctly. In some places the ganglion cells have wholly disappeared.

These, then, are the pathological minute lesions in Bright's disease in the ganglia regulating the heart movements. Before entering into the question of the bearing of the abnormal appearances, it may be well to answer the question: How does death come in the hypertrophied heart of Bright's disease? It comes with contraction of the left ventricle and dilatation of the right; in other words, it is a death of apnœa rather than one of asthenia. The diseased ganglia do not produce paralysis of the heart. As an instance of how even acute destruction of the ganglia will cause death, similar, as regards the heart, to that in Bright's disease, I cite the following case of injury and of hemorrhage into the cervical ganglia, the autopsy of which was most carefully made by Dr. Longstreth.

Thomas McC——, aged fifty, admitted into the Pennsylvania Hospital, February 1, 1882. The patient fell on shipboard, fractured the third, fourth, and fifth cervical vertebræ, and became immediately paralyzed, both as to motion and sensation, from the level of the second rib and deltoids downward; breathing was diaphragmatic; the pupils were contracted. There was priapism and retention of urine; the heart's action was frequent and feeble. Post-mortem examination showed large clots pressing on the cervical ganglia; at the third vertebra the cord was compressed and softened. The lungs were congested; the right heart was distended, the left ventricle firmly contracted. The microscopic examination of the ganglia, especially the inferior cervical ganglion, exhibited in many places the presence of extravasated blood around the ganglion cells; some of these cells showed already changes, especially pigmentation, although but a few hours had elapsed between the accident and death; in other respects the cells and tissue of the ganglion appeared normal. The kidneys were normal.

But to return to the examination of hypertrophy of the heart in Bright's disease. I have, I trust, made clear to you that there exist, associated with the enlarged heart, marked changes in the ganglia of the sympathetic nerve, from which the cardiac nerves are derived. Is it going too far to assume that these changes are an integral part of the disease; that they may determine the hypertrophy?

How, I cannot explain to you exactly. It would not be difficult to bring forward a theory of loss of controlling influence on the heart, produced by the altered condition of the nervous apparatus; but our knowledge of the physiology of the nerve supply of the heart is not accurate enough to admit of absolute reasoning from cause to effect. Still, that disturbed nerve-power produces changes in structure by affecting its nutrition, we know, in every tissue of the body. Why should not the same be true for the heart? Again, if we adopt this view of the derangement of nerve condition and of the vaso-motor influence being the starting-point, we can readily explain the well-known lesions in the vessels, both large and small. Indeed, when we look at the wide-spread changes which occur in them in Bright's disease, especially in its chronic form; when we see everywhere accumulating the evidence that these changes happen in all the vessels of the body—in those of the spinal cord, the brain, the lungs, the liver, in veins as well as arteries—we are driven to the conclusion that there must be some general cause at work which determines so great, so universal an alteration. That general cause, from the nature of things, can only be in the blood, in the vascular system itself, or come from the nervous offices which give life to the vascular system. The change is too extensive to be a mere self-determined local growth of the coats of the vessels. We cannot, indeed, ignore the agency of the waste-laden blood in its full development. But what starts the change? What is the cause of the degeneration? Is it not fair to look, beside the blood, to a cause so predominant as the nervous influence, which is everywhere?

I would thus locate as most likely the original starting-point of the alteration, alike of the vessels and of the heart muscle, in the nervous ganglia and in the parts of the nervous system controlling the nutrition of these textures. What the ultimate cause of the lesion is cannot be stated, nor need we assume that one cause alone will determine it. It may be gout, it may be lithæmia, it may be rheumatism, it may be alcohol, it may be lead, it may be mere purely perverted nervous function from worry, from strain, from anxiety. Any one of these causes may start that alteration in the ganglionic nervous system which leads to degeneration and to the formation of increased fibrous tissue and subsequent atrophy to, and with it also the heart, and it is that change in the vascular textures supplied by the derangement of the heart and affected ganglia and nerve-filaments.

These are, of course, not matters that can be at once proved or adopted. You must take them as lines of thought which have come naturally from the investigations

pursued, and which promise as rich a return as any that we have had from following out views hitherto brought forward. Whether the suggestions based on these researches prove to be correct or not, I think that the observations themselves furnish one more proof of the general character of what we call Bright's disease, especially in the cirrhotic form. They show us that the primary changes are to be looked for away from the kidneys, not in them, and that there is a general disease, based on a general wide-spread cause, underlying the vascular changes, heart atrophy, and kidney disease.

But to return from these reflections to the state of the heart in Bright's disease. Looking now at the result of all the observations and the process of reasoning by which I have endeavored to collate them; looking at the general pathological facts which have been cited, I draw this conclusion: That the cardiac hypertrophy which is found in Bright's disease is not in any sense the consequence of that disease. It is an integral part of the same general morbid process, of which the kidney lesion is only the most obvious expression. You may believe, with me, that it is in the nervous system that we are to look for the ultimate, or, it may be more correct to say, the original source of the malady. You may hold that this is not proven, that the changes there are only part of the general changes described, and are not the first; that they are result, not a cause. But I trust you will see how disseminated an alteration they indicate; how everything points to a general rather than to a local malady, and not think me too venturesome in stating that chronic Bright's disease ought not to be looked upon as an affection of the kidney at all, though it is there that, to the naked eye, its most pronounced changes occur. In adopting this conclusion, the cases that die of apoplexy from disease of the vessels of the brain before the kidney disease shows any signs; the cases with general vascular tension in which there is as yet no albumen or other signs of renal malady; the cases with marked, the cases without as marked, cardiac lesion, receive their ready explanation. They cease to be objects of wonder. The mischief wrought by the general malady has been wrought more elsewhere than in the kidneys. It may exist to a greater, it may exist to a less degree in any structure; it may be diversified in a very varying degree, and be present in parts in very alterable proportions.

These conclusions I draw more particularly from a study of the pathology of the contracted kidney. Perhaps it would be better to limit them to this condition, and to call this general malady Bright's disease of the kidney only until someone with a genius for names gives it one we can all adopt. There may, in fact, be some of the forms of

the so-called Bright's disease which are really affections of the kidney; there may be deposits or degenerative processes in the kidney simply superadded to the textural changes in the contracted organ. These intricate questions cannot be here discussed; they are, indeed, with present knowledge, beyond our solution. But, examined in the light of the view which we have inquired into to-night; with each group of cases looked at by itself; with the structural alterations in other organs and parts than the kidneys, minutely inspected; with the condition of the blood-vessels everywhere, and of the nervous ganglia, noted; with the antecedent changes as far as possible considered, it may be then that other groups will stand forth separately as showing diverse general states as their basis.

No pathological research in these days of keen induction stands long by itself. No view can be even approximately proved, that we do not at once begin to search how, as practical physicians, we can make it available for the prevention or cure of disease. That the treatment of Bright's disease, and of what are regarded as its complications, is not what we wish for, is everywhere felt. If lines of investigation and of thought should lead to the recognition of the malady being a general rather than a local one, and discern its starting-point, lines of practice which are in accordance will be pursued, and it is by following these lines that helpful and hopeful results will, I believe, come. As yet, even with the wish to work in that direction, we may not have the exact agents for the purpose. But science now is too resolute to halt helplessly before obstacles when once the path beyond is clearly recognized.

In Memoriam.

MIDDLETON GOLDSMITH, M.D., LL.D.

A BIOGRAPHICAL SKETCH OF THE LATE MIDDLETON GOLDSMITH, M.D., LL.D.

By JOHN C. PETERS, M.D.

Dr. Goldsmith only lived in New York from 1837 to 1845, but during that time he became one of the founders of this Society. Most of the rest of his life was spent in Vermont. Few of our members had ever seen him, and still fewer have any clear conception of the man. Yet his life was a very picturesque one, crowded with good works, and crowned with great successes. His professional renown commenced very early, for he was a good anatomist and almost a great surgeon while yet in his teens. He commenced to dissect when some of his boyish companions were still whittling sticks. His father, Dr. Alban Goldsmith, was Professor of Surgery in the Louisville, Ky., Medical School, and Middleton had served as his prosector and clinical and surgical assistant, and performed at least one amputation of the thigh before he was eighteen years old. He always seemed to have anatomy and surgery at his fingers' ends.

In 1837, when he was nineteen years old, his father was appointed Lecturer on Surgery in the New York College of Physicians and Surgeons, then in Crosby Street, and Middleton, of course, continued to assist him. This college was then the only medical school in New York, and in 1837, when I first sat upon its benches, it had less than seventy medical students. Philadelphia then had four times as many medical colleges, and more than ten times as many matriculants. Now the disparity is not so great. Middleton graduated from the College of Physicians and Surgeons in 1840, aged twenty-two, and almost immediately sailed to India and China, as a ship's surgeon.

On his return he established, with the aid of Drs. Markoe, Sayre, La Conte, and others, the first Alumni Association of the College of Physicians and Surgeons, and contributed largely to the first volume of its Transactions.

The Goldsmiths, father and son, were great naturalists, and very fond of rare and handsome birds; hence Middleton was soon found assisting the great Audubon in

the dissection and classification of some of "the Birds of America." They had many artistic and æsthetic tastes, and their beautiful rooms in Park Place were filled with handsome illustrated books, fine engravings and paintings, splendid cameos and musical instruments, besides being loaded down with brilliant surgical apparatus. There were then no stores in Park Place, or on Broadway, near by. These wide streets were filled with stylish private residences, and the great Park on the east, and the lovely Columbia College grounds on the west, made the whole neighborhood delightful.

But the Goldsmiths came, on one side, from "a race half aristocratic and half bucolic; as simple as shepherds and as proud as kings;" hence Middleton was often found at Bull's Head in search of tape-worms, hydatids, liver flukes, and other curious pests of animals. He also made many minute dissections of the eyes of men and animals for a prominent oculist, and even taught him how to operate. He was a great favorite with the genial but eccentric Dr. John W. Francis, some of whose peculiarities he may have imbibed. Dr. Francis always spoke of him as the last lineal descendant of the great Oliver, and as a poor medical orphan, who had no uncles in the New York or Bellevue Hospitals to help him on. In return Middleton poked great fun at the triplex and complex pills, and the big and little worms, gall-stones, and bilious disorders which Dr. Francis believed they cured.

He visited the dead-house in the Park every day to make autopsies for the coroner, and, when there were no other subjects, he and I cut up the bodies of the paupers, which were merely sent there for burial in Potter's Field, which used to be in Washington Square. I well remember his long and swift, but skilful, way of cutting; the quick hari-kari-like, or "happy-dispatch" way with which he went through a cadaver, without any rough mangling or jumbling of things. All was orderly and rather artistic.

At this time he was a remarkably tall man, considerably over six feet high, muscular, but not gross, and exceedingly well-proportioned. He was quick, but easy and even graceful in most of his gestures and movements. He had reddish hair, a fair complexion, a handsome and pleasant face, and was very neat and even showy in his dress. He was a reasonably good Greek and Latin scholar, but especially a perfect master of the English language, which he always spoke and wrote with great purity and conciseness. He was very fond of Burns and Shakespeare, and that grand old pagan, Hesiod, whose "Works and Days" and "Theogony; or, Creation of the World and Gods," he delighted in all the days of his life. He sometimes made verses, which he called poetry; always

sang a good song, and sometimes a better psalm tune; he could play on the guitar and banjo; was a good talker, a capital story-teller, and quick at repartee. He soon became a great favorite in medical, social, and religious circles, and often shone in the best assemblages of the brightest men and nicest women. He was looked upon as a sort of modern Apollo and Admirable Crichton, and the most rising young surgeon in New York. But he blazed in his greatest glory when he paraded as Brigade Surgeon on Major General Sandford's showy militia staff. He was always gallantly mounted, for he was born in Maryland, reared in Virginia, and trained in Kentucky, and knew what a good horse was. Dr. Sayre and I often gazed at him with admiring, but also envious, eyes. How different was his composed and stately bearing from the grotesque performances of Regimental Surgeon Paine, who always appeared on "Training Day," crowned with an old revolutionary cocked hat, with a big red feather in it; armed with an ancient cutlass buckled over his every-day frock-coat; and mounted on a vicious little Vermont pony, which pranced, and kicked, and buck-jumped in such outrageous fashion that Martyn sometimes seemed seated on his horse's ears and sometimes nearer the ground.

Middleton occasionally mounted a metaphysical high horse, and used involved language, so tumid as to be scarcely intelligible, but which immensely tickled Dr. Sayre, and would have greatly pleased a High Dutch Professor of Transcendental Philosophy. He also always had a great deal of what Hughlings Jackson calls "mental diplopia," *i.e.*, he could look equally earnestly and impartially on both sides of a question, and could not only defend each view with the greatest ingenuity, but with the most perfect good faith and honesty.

In those times French pathology was in the ascendant, and the Goldsmiths and many other New York physicians were worthy disciples of Cruveilhier, Andral, Louis, Civiale, etc. I had been educated, in part, in Germany, and was filled with enthusiasm for Rokitansky and the Vienna school of pathology. Dr. Goldsmith and I often talked over pathological matters in the dead-house and elsewhere. He wished me to send to Vienna for barrelfuls of specimens, and to found a pathological society, of which none then existed in the world. Dr. Sayre also wanted to establish a pathological society, and soon I was invited to attend a preliminary meeting for this purpose at Dr. Sayre's office, and am said to have made my appearance with two pailfuls of specimens, collected by Dr. Goldsmith and myself. Middleton preferred the plan of the Boston Society for Medical Improvement, which was partly clinical and partly pathological. Dr. Sayre had a

plan for a purely pathological society, and he and Dr. Goldsmith persuaded those ripe pathologists, Dr. John A. Swett, John A. Forry, Thomas M. Markoe, Gustavus A. Sabine, Robert Watts, William C. Roberts, and others to join them; and soon Willard Parker, Alonzo Clark, John T. Metcalfe, William H. Van Beuren, and others of like stamp were added to the number and quickly overshadowed the three original suggestors and founders of this society, which undoubtedly were Drs. Goldsmith, Sayre, and J. C. Peters.

The first meeting took place on June 14, 1844, and in less than six months Dr. Goldsmith was called to the Chair of Surgery in the Castleton, (Vt.) Medical College. He never returned permanently to New York, but never forgot the New York Pathological Society, which he regarded as a bantling of his own, and finally made a generous bequest to it to increase its honor and usefulness.

In Castleton Dr. Goldsmith lectured and operated for ten years, making a great reputation and gaining many friends, most of whom clung to him during the rest of his life. Dr. Sayre won a prosectorship, and fell heir to the Brigade Surgeoncy and to the magnificent uniform, which he had to let out over the chest and elsewhere; for although he was not too large for his own trousers, he was for Goldsmith's.

In 1851 Middleton was chosen President of the Vermont Medical Society, after only six years' residence in the State, and made a capital report to it on fractures.

In 1856 he was called to Louisville, Ky., to the chair of surgery which his father had filled before him, and soon became Dean of the Faculty. He was summoned occasionally to New Orleans, St. Louis, and other distant places in consultation and to operate. Sometimes he got a barrelful or less of rare old whiskey, as well as thousand dollar or smaller fees from G. P.'s, or grateful patients; but he always preferred hard cash to hard liquor. He often had to treat what Shakespeare called "the rotten diseases of the south, such as gripes, ruptures, fistulas, gravel in the back, stone in the bladder, big livers, and aching bones;" but he was specially noted for his skill in operating on the eye, and for successfully crushing stones in the bladder.

When the civil war broke out he promptly offered his services to the Union, and commenced as Brigade Surgeon in the Army of the Cumberland in 1861. Soon he was Medical Director in General Buell's army at Shiloh; next, Inspector of Hospitals in General Grant's army at Corinth; and, finally, Surgeon-in-Chief of all the military hospitals in Kentucky and the Army of the Ohio. It is hard to overestimate the good work which he did in

these honorable and important positions. In addition, he discovered the virtues of bromine in hospital gangrene and kindred diseases. He must be regarded as one of the great pioneers of disinfection, as Lister did not commence his experiments till 1866, and little was known about carbolic acid before 1870. When his sick and wounded were dying for want of warm clothing, he disinfected condemned blankets and overcoats, which had been used by small-pox patients, with bromine, and succeeded so well that he not only used these clothes himself, but permitted his whole family to do so also.

He now broke down completely under a severe and prolonged attack of camp diarrhœa, but persisted enthusiastically in working up his views on bromine. His attractive daughters had many suitors, whom he quickly pressed into the dreary task of copying papers on gangrene, pyæmia, etc. As none of them were medical men they naturally thought they had fallen on hard lines indeed.

At the close of the war he endeavored to resume his practice in Louisville, but his outspoken Union sentiments rendered him so unpopular that he was forced to return to Vermont, where he remained, in Rutland, for the last twenty-five years of his life. He did not again enter into general practice, but played a large rôle in surgery, as a consulting physician in obscure and difficult cases, and as an expert witness in medico-legal trials. He soon again became a power in Vermont. He established the Rutland Free Dispensary, and was ever ready with his purse and services. There is no part of his history which is more clear and creditable to him than his devotion to the poor. He not only treated those who came to him, but sought out many who could not easily get other medical or surgical aid; and generally also paid their board bills. He was often crusty and abrupt to the rich, and is said never to have yielded to the whims of his patients unless they were very poor, or very old, or in some way peculiarly unfortunate. On alternate Sundays he would drive far out to the marble quarries and prescribe for all the women and children of the Irish laborers. On other Sabbaths he would go to the wood-choppers' camp for the same purpose. He never exacted fees for these services unless the men were careless or brutal to their families. Then he not only charged high, but made an effective use of language. Some of the laborers would slip their silver dollars into the big pockets of his huge shooting-jacket, where they would often be found long afterward, mixed up with cartridges, prize potatoes, and packages of fish-hooks. He had as much influence with this class of people as their priests and clergymen; and was a great friend and chum of good Catholic priests, kindly Congregationalists,

jolly, shouting Methodists, quaint hard-shell Baptists, and gentlemanly low church Episcopalians; but he looked askew at canters and ranters and bitter Calvinists, and had rather a disrespect for his Satanic Majesty, and for his brimstone and the place where he is said to burn it. As the New England Sabbath always ended on Sunday afternoon, he generally had a gun and fishing-rod quietly stowed away in his wagon.

He delighted in assisting young doctors by good counsel, by speaking a kind word for them where it would tell, by helping them in their operations, and by lending them medical books and surgical instruments, many of which he never got back again. Still, he always had the largest medical library in Vermont, and the greatest collection of surgical instruments, besides several big galvanic batteries and unusually fine microscopes; all of which he knew how to use.

Although very fond of his professional work, he steadily began to delight more and more in out-door pursuits and pleasures, in fine horses, cattle, sheep, dogs, guns, farming and gardening, or as the old geographies put it, in "hunting, fishing, and war." He became a sportsman and naturalist in the best sense of those terms, for there was not a plant or flower, or seed or grain, or bird or fish or animal that he did not know all about.

He always had great respect for

> That germ, but faintly understood,
> Whose growth is hid in motherhood,

and hence engaged in breeding fine horses, cattle, sheep, dogs and even fish, and helped to make Vermont a great cattle and sheep-raising State. Next he had model farms and raised choice grain, fruit, flowers, and potatoes. He even tried hard to make Vermont cider as attractive as sour Rhine wine. In short there was scarcely any branch of useful domestic knowledge with which he was not familiar. He never failed to master any subject in a comparatively short time, and whatever he studied soon yielded good practical results to some one. He required ready outflows for his surplus energy and always selected agreeable or beneficent ones. His surgical work was like second nature to him, as he had been operating largely for thirty years or more. In short he had long reached that highest and best development of his faculties when they had become, as Schiller says, almost automatic without ceasing to be either accurate or earnest. As his bucolic side predominated more and more over his aristocratic tendencies, so did he shed his conventional and professional clothes and dress more and more like a prosperous farmer and woodsman. He could not and said he would not always look like a first-

class undertaker or immaculate head waiter. His broad-brimmed stiff hat, which was both sunshade and umbrella, could be seen from far off, and there was no other in Vermont like it; his boots were apt to be high and water-proof, for he never knew when he would wade for trout, or plod for partridges, and his coats were often like large shooting jackets, or a collection of game bags. On week days he often had a long bamboo fishing-pole sticking out behind his wagon, but he drove fleet horses and was rarely dusted on the road.

He was generally provided with a medicine chest and case of surgical instruments, was always ready for any emergency, and came into the sick room like a wholesome and cheery breeze of wind on a sultry day. He had no favorite operations, but was always called upon when unusual skill was required, and is credited with the usual prodigious number of big tumors, aneurisms, amputations, and of operating on all sorts of strangely visited people, the despair of other surgeons. It is even said that he would not even leave a mole, or mother's mark, or hare lip, or ugly scar to disfigure fair women, or pleasant children.

As an expert witness he was without a rival; a terror to opposing lawyers, whose cross-examinations he always turned against themselves, and is said never to have been defeated. His commanding presence was so impressive and his testimony was so clear, concise, and positive that the jury always believed that only they and he understood the case rightly, and if necessary would have over-ruled the judge and the laws; but he always obtained the approbation of Chief Justices Peck and Shaw, Judges Phelps and Barrett, Governor Stewart and other great and honest lawyers.

As a philanthropist and hygienist, he was a great thorn in the sides of ignorant or lazy health officers. He found the Vermont State Asylum crowded with over five-hundred inmates when there was only room for one-hundred and fifty. Besides, the State was only allowing $1.40 a week for board which cost nearly $3, and was not even paying that poor pittance, as it was $30,000 in arrears. We can easily imagine him saying to the Governor and Legislators: "If your excellencies will not merely put your venerated noses in these overcrowded rooms at midnight, but will endeavor to sleep the rest of the night in them, you will find out all about foul air, and what we doctors call lack of ventilation. If you will ask your thrifty wives to feed and clothe these poor patients on the scanty State allowance they will soon give you lessons in domestic economy that you will never forget, or else I have strangely underrated the generosity and justice of Vermont women."

He drew up the game laws of Vermont, which have been the models for those of several other states, making sportsmen and hunters the wise and generous protectors of game in the breeding season. Then poachers and pot-hunters had no rights which any decent man was bound to respect. When fish and birds and wild animals were mature, swift, cunning and strong, he could whip a stream, snare birds, and shoot beasts with the best of them; then he almost rivalled the poachers, especially when he wanted delicate food for poor patients. His game laws were of course attacked, but it is shrewedly suspected that the most astute and bitter criticisms came from his own pen anonymously, and that he greatly enjoyed his own apparent discomfiture.

Need we be surprised that when such a man died there was a great outburst of overwhelming sorrow from his neighbors, and that his distant friends were almost astounded at the tremendous praise which was heaped upon his memory. But when we find that even his mother-in-law, and his brothers-in-law, and sons-in-law, joined heartily in this praise, we must believe it well bestowed.

We can never forget him. Several years ago he gave this society a large sum of money to found a lectureship; for many months he had been endeavoring to give us his large and valuable medical library, estimated to contain 6,000 volumes. Then his dear wife, whom he did not expect to outlive, was suddenly carried away to a better world, and he was stricken down with deadly grief, helpless and unconscious from which he only aroused sufficiently to complete his generous gifts to us and to the Academy of Medicine.

Oliver Goldsmith wrote: "Innocently to amuse the imagination in this dream of life, is wisdom." Middleton Goldsmith acted as if he might have said: "To usefully and generously train our brains and our hands and our hearts in this royal game of life is still greater wisdom."

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